DELAWARE STATE MEDICAL JOURNAL

Official Organ of the Medical Society of Delaware

INCORPORATED 1789

VOLUME 25
NUMBER 4

APRIL, 1953

Per Year, $4.00
Per Copy 50c

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Entered as second-class matter June 28, 1929, at the Post Office at Wilmington, Delaware, under the Act of March 3, 1879. Editorial Office, 822 North American Building, Wilmington 7, Delaware. Business Office, Farnhurst, Delaware. Issued monthly.

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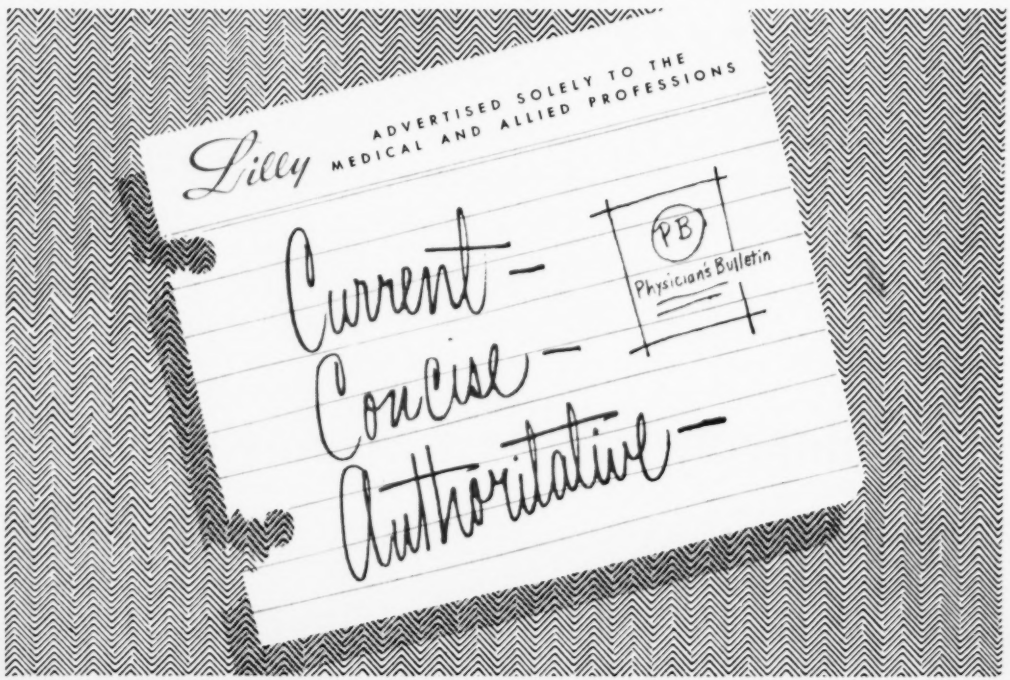

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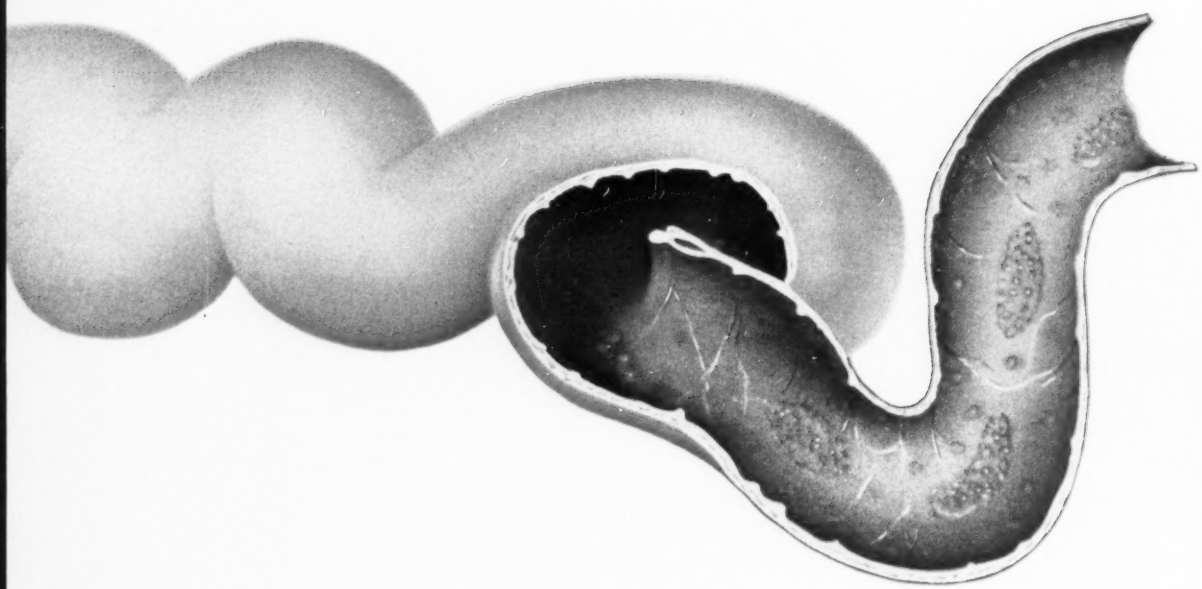

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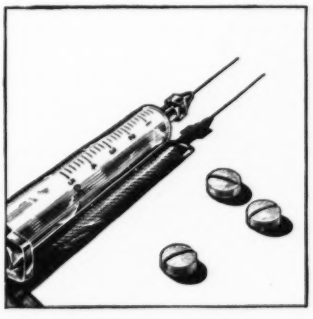

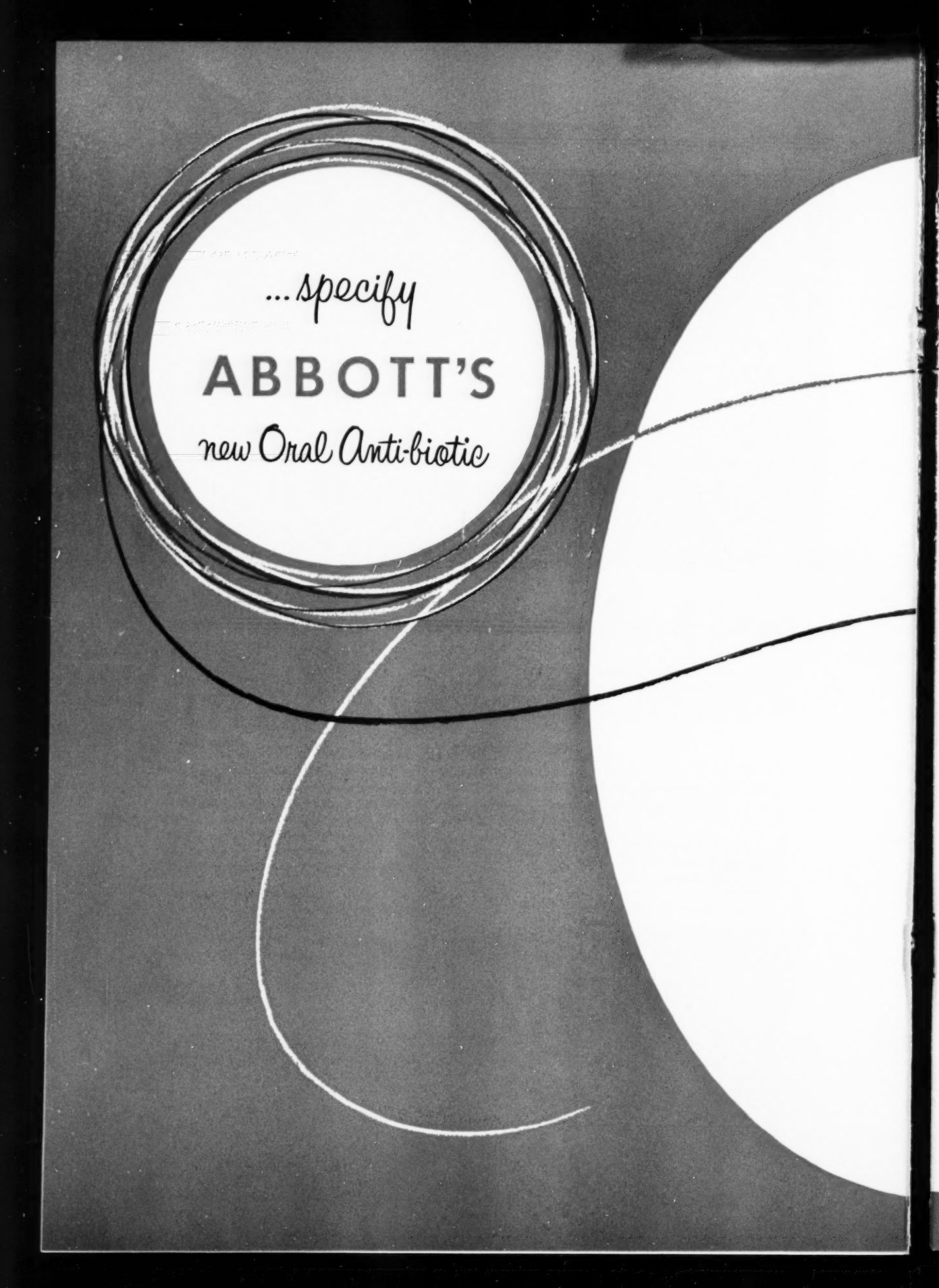
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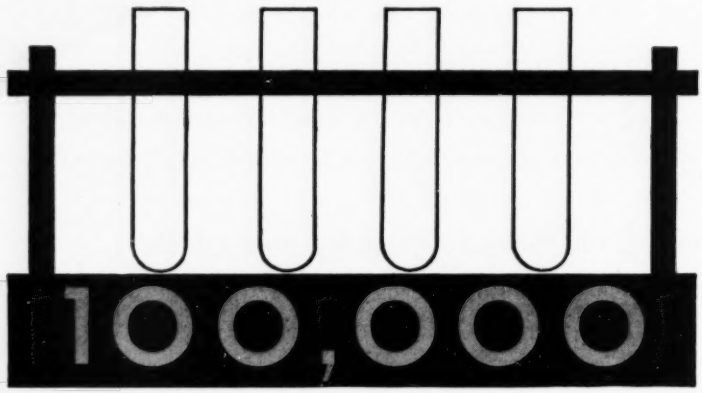
1. McGuire et al. (1952), J. Antibiotics & Chemo., 2:281, June.
2. Heilman et al. (1952), Proc. Staff Meet. Mayo Clin., 27:385, July 16.
3. Haight and Finland (1952), New Eng. J. Med., 247:227, Aug. 14.



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1. Blotner, H., and Marble, A.: New England J. Med. *245*:567 (Oct. 11) 1951.
2. Getting, V. A., and others: Diabetes *1*:194, 1952.
3. Wilkerson, H. L. C., and Krall, L. P.: J.A.M.A. *135*:209 (Sept. 27) 1947.

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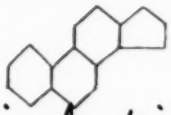
the symbol SCHERING denotes leadership in steroid hormone  research and manufacture -- including Cortogen in the most useful clinical forms. For systemic therapy, use Cortogen tablets SCHERING or injection . For eye disorders use Cortogen ophthalmic suspension -- sterile . The Schering seal SCHERING appears on each tablet and package -- your assurance of quality.

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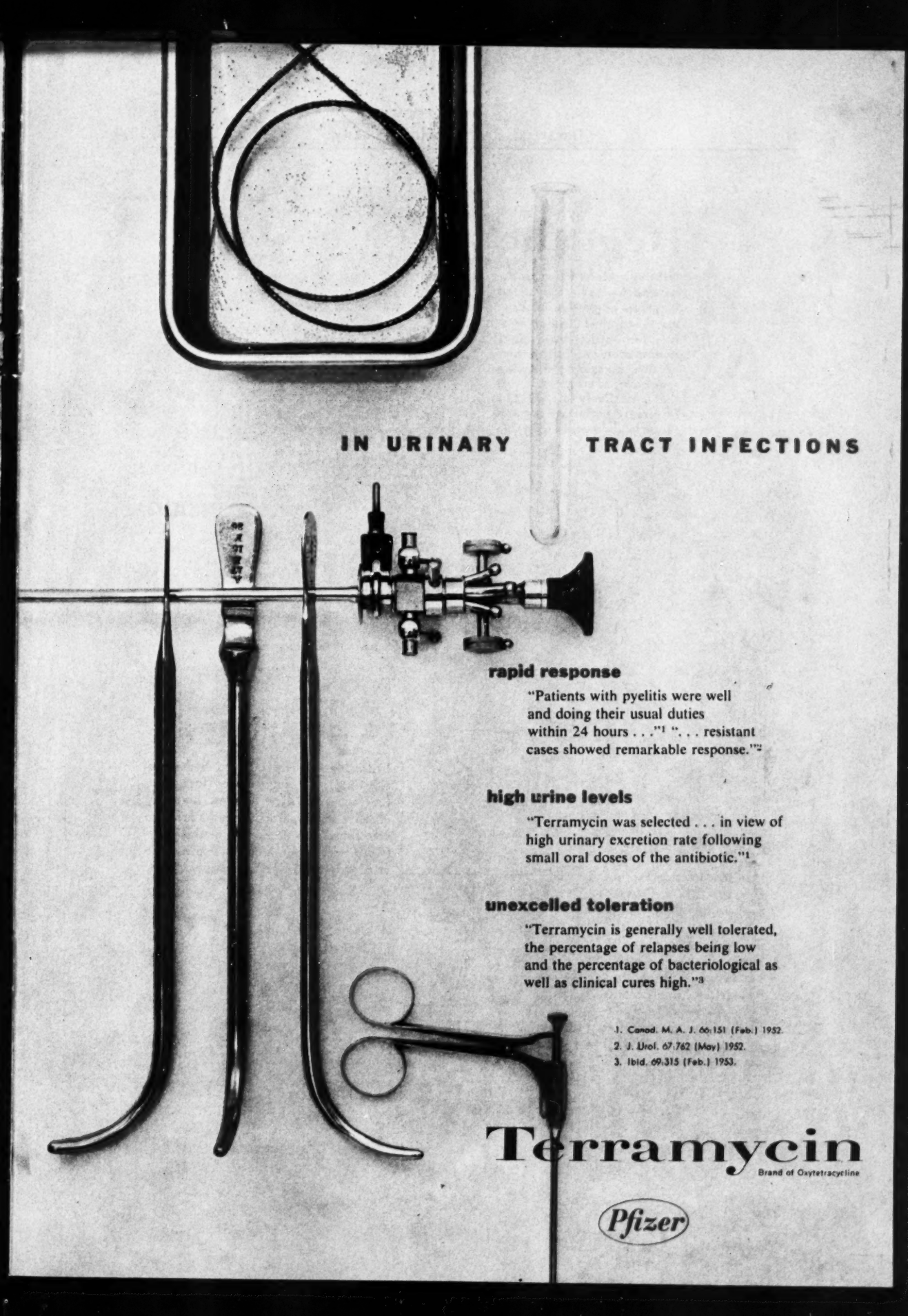

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1. Canad. M. A. J. 66:151 (Feb.) 1952.
2. J. Urol. 67:762 (May) 1952.
3. Ibid. 69:315 (Feb.) 1953.

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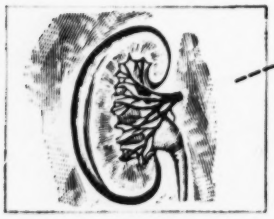

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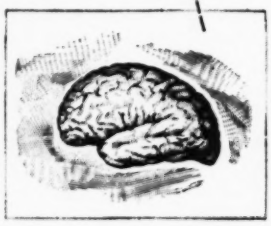

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2. Superficial temporal artery & vein
3. Temporal branch of facial nerve
4. External carotid artery & posterior facial vein
5. Superficial cervical lymph nodes
6. External jugular vein
7. Accessory nerve & internal carotid artery
8. Platysma muscle
9. Fourth cervical nerve
10. Superior position of sternocleidomastoid muscle
11. Deep cervical lymph nodes
12. Fifth cervical nerve
13. Posterior supraclavicular nerve & anterior jugular vein
14. Superficial cervical artery & vein
15. Middle supraclavicular nerve & subclavian artery
16. Transverse scapular artery & vein
17. Inferior position of sternocleidomastoid muscle
18. External maxillary artery & anterior facial vein
19. Submaxillary lymph nodes & digastric muscle
20. Submaxillary gland & mylohyoid muscle
21. Submental lymph nodes & hypoglossal nerve
22. Superior laryngeal artery & nerve
23. Superior cervical ganglion
24. Superior laryngeal vein & omohyoid muscle
25. Superior thyroid artery & vein
26. Ansa hypoglossi
27. Common carotid artery & sternothyroid muscle
28. Middle cervical ganglion & phrenic nerve
29. Vagus nerve
30. Thyroid gland & middle thyroid vein
31. Internal jugular vein
32. Sternohyoid muscle
33. Jugular lymphatic trunk
34. Inferior thyroid veins

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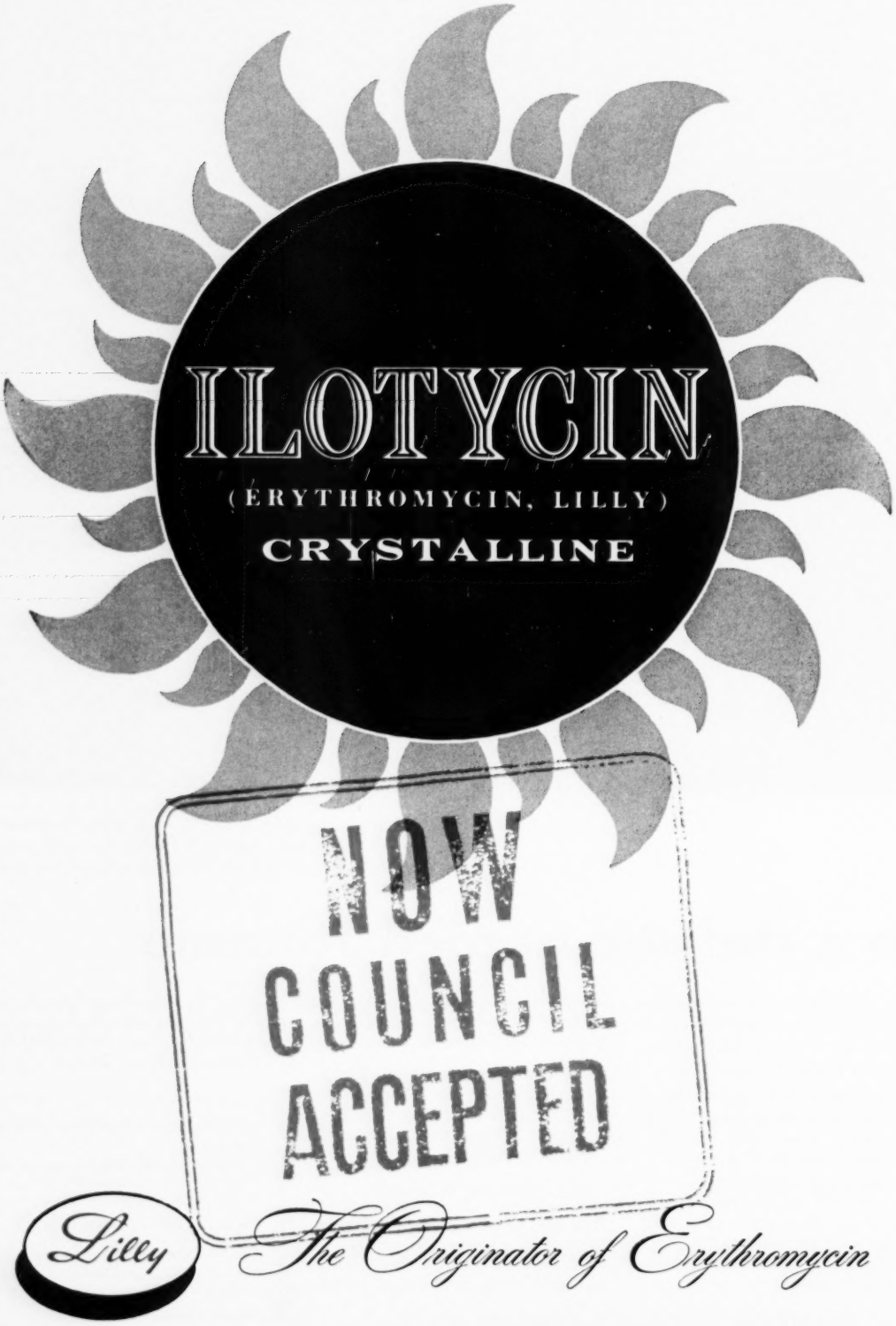
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APRIL, 1953

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THE HERNIA PROBLEM
Confessions and Impressions

JOHN C. PIERSON, M. D.,*
Wilmington, Del.

Even the casual reader of surgical journals cannot help but be impressed by the frequency with which the subject of hernia rears its ugly head. The adjective "ugly" will not seem inappropriate to those who have had a reasonably active experience with the problem of hernia. They will agree, I think, that even though herniorrhaphy was one of the earliest surgical procedures attempted, and continues to be performed in great numbers each year, a full and complete satisfaction with results in this field has not yet been achieved. In spite of the profusion of papers dealing with various aspects of the problem one finds that a discouraging if not actually chaotic state of misunderstanding still exists. Detailed anatomy of the inguinal canal, even the lack of uniformity in terminology, operative techniques, the incidence of recurrence and the factors responsible for recurrence—these points and others need only to be mentioned to illustrate the complexity of the problem. The Smith-Petersen nail has done much to invalidate Kellogg Speed's designation of the femoral neck fracture as the "unsolved fracture," but to date no such magic wand has come to the rescue of the hernia problem.

In the face of these facts it is with some temerity that I embark upon further discussion of this subject. My sole reasons for such a venture are first that the problem itself is a fascinating one, and secondly that the points to be brought out are gathered from the bibliography of personal experience and may not have been sufficiently emphasized previously. There is something less exotic about the repair of a hernia than one finds in a major gastric or intestinal resection for an extensive neoplasm. There is also a world of difference in one's reaction to that post-operative bugaboo peculiar to each, viz., recurrence. Chagrin is a mild word for the one; disappointment amply describes the other.

What then has my own experience with the hernia problem taught me?

DIAGNOSIS

While only occasionally does one encounter difficulty in detecting the presence or absence of a hernia, now and again the question will try one's skill in this field of examination. It is probably true that a patient is more often tagged with the diagnosis of hernia when none actually exists than that error in the reverse direction is made. Some of the reasons for the "false positives," it seems to me, are (1) that a patulous or dilated external inguinal ring is construed as a hernia per se; (2) the failure to understand what actually constitutes a cough impulse; (3) muscle bulge. Muscle bulge is seen in thin, muscular individuals and in no sense represents a hernia. It does not require operation, and in fact cannot be materially altered by a plastic musculo-fascial maneuver. Muscle bulge, as distinguished from true hernia, is always bilateral and is characterized by a ballooning of the internal oblique muscle, not so much in the canal as lateral to it. There is no true cough impulse.

On the "false negative" side one may sometimes find it extremely difficult to demonstrate a hernia of the indirect type even though the history seems unequivocal. This is especially true in the case of children and in patients who have been wearing a truss right up to the time of examination. It occurs also in a small group of patients who though not wearing a truss give a history that only very occasionally does a "lump appear in the groin." In this type of patient the internal ring is small, the sac almost obliterated, and only when omentum or

*Director, Department of Surgery, Memorial Hospital.

bowel finds it way through the abdominal ring and distends the sac can a positive finding of hernia be established.

It is true of course that incarcerated omentum in the sac of a femoral hernia may pose a problem since the hernia will not give a cough impulse and being non-reducible may be confused with an enlarged lymph node or a soft tissue tumor.

In the field of differential diagnosis there arises also the desirability of pre-operative recognition of indirect as opposed to direct hernia. My batting average in this respect is only fair. It is not purely an academic matter, however, since in the case of indirect hernia, one can offer a much more certain prospect of permanent cure, even though such a patient is obese, in the decade of life described as the youth of old age, and whose daily habits involve strenuous physical effort. I believe that one should try in his diagnostic examination to arrive at a definite impression as to the type of hernia present and should so designate it on his office record.

AGE

While it is undoubtedly safe to operate upon infants at any age and in the case of incarceration, one has no other choice, in general it is my policy to defer operation until the child is around 2-3 years of age. At this age the cord and other structures are not so tiny, urinary incontinence is less of a problem and the general care of the patient seems easier. When, however, there is a history of recurring episodes of irreducibility or evidence that a yarn type of truss fails to retain the hernia, I believe that nothing is gained by waiting until an arbitrary age of 2-3 years is reached. It has been my observation that few instances of permanent cure of hernia in children can be expected through use of a truss alone. Many such cases apparently "cured" at 5-6 years of age will "develop" a hernia in later years (12-25).

At the other extreme of life my position is even less equivocal. I feel that any patient *having symptoms* related to his or her hernia—whether that patient is sixty, seventy or even eighty years of age—should be offered a surgical repair of his hernia. If necessary, operation can be done under local anaesthesia. One has only to be the recipient of a smile of gratitude from an elderly patient who has been able to discard a poorly fitting, irritating, and ineffectual truss to be convinced that when a patient's hernia is a source of annoyance, it is a kindness to offer him the alternative of operation. This is especially true in the large scrotal type, even though there is no history of periodic incarceration. A small, symptomless, direct protrusion in an elderly patient is not considered an indication for operation.

TRUSS

I have little to say in this regard beyond the fact that if a truss is to be used, its proper fitting should be checked by one thoroughly familiar with the anatomy of the inguinal canal. Furthermore, I can find no reason for using a double truss if the hernia in a given case is demonstrable on only one side. A truss finds its greatest usefulness in the small direct type of hernia and is a futile gesture in the complete (scrotal) type of indirect hernia.

TECHNIQUE OF REPAIR

This is a wide and fertile field for both academic and practical discussion, but it is quite beyond the intent of this paper.

As a fundamental principle, I believe that the operating surgeon should be prepared to vary his approach according to the circumstances peculiar to the case in hand. On this basis he will find it expedient to run the gamut of such modifications as orchiectomy, the use of tantalum mesh, fascia lata, perhaps a cutis graft, etr. Certainly it is true that in my concept of hernia repair, proper re-enforcement at the point of critical weakness is worth more than all the added steps to buttress the first line of defense. A few properly placed sutures of whatever material, but grasping tissue of good fascial quality, and avoiding tension give one the same sense of security that observance of similar principles gives to closure of the duodenal stump as part of a gastrectomy.

Today most surgeons prefer non-absorbable suture material—silk, cotton, or wire—and some claim a lowered recurrence rate as a corollary to this practice. I happen to prefer silk or cotton, and yet I must confess

that in the occasional case complicated by prolonged "spitting" of this material, I have wished that I had stuck to my former habit of using catgut.

The question sometimes arises as to whether appendectomy or other procedures should be added to the primary hernia repair. I have found no reason to avoid such complementary operations where the viscus is accessible and not involved in an acute process that might contaiminate the hernia wound.

Another controversial point has to do with the simultaneous repair of a bilateral hernia. Recurrence under such circumstances is not merely a matter of subjecting the patient to a second operation and therefore no more taxing on the patient than if the original decision had been in favor of a two-stage procedure. As has been well said, the most difficult kind of hernia to repair is the recurrent hernia. In spite of personal experience with recurrence where bilateral repair has been attempted in one stage, I have not found it practical to follow any routine in this regard. Rather the decision to proceed with the opposite side is based upon the findings encountered on the side first approached. The side having the larger defect is selected for the first operation, and if the tissues are found easily susceptible to re-enforcement without undue tension and without need of supplementary material, the second side is undertaken at the same sitting.

Another point that might be made in respect to operative technique has to do with disposition of the sac in cases of direct hernia. It is my contention that since in such cases the neck of the sac is not narrower than the fundus, nothing is gained by isolation and subsequent ligation of it. Effective repair of this type of hernia is dependent rather upon reconstruction of the canal floor itself. For the last several years in attempting to accomplish this end, I seem to have become enamored of the McVay modification, making use of Cooper's ligament as a firm unyielding anchorage site. Anatomically and physiologically this maneuver seems to make sense. It is readily admitted that all operators do not subscribe to this view, but current writings would seem to indicate that the McVay method of repair has won many converts.

Specific Technical Considerations

Under this caption I would like to refer to a few details that in my own experience seem to have acquired special importance and value.

1. Of these I would mention first the desirability of preserving the umbilical cicatrix in repair of hernias in this region. This is easily accomplished by a curved incision either at the upper or lower rim of the umbilicus and reflection of the skin flap accordingly. Just prior to skin closure one or two sutures tacking the dermal layer to the underlying fascia will umbilicate the skin and effect a very natural restoration of this particular anatomic landmark. Some may not agree that the cosmetic objective described deserves the attention accorded it here, but some experience with it convinces me that it is a worthwhile refinement of the standard approach.

2. Repair of inguinal hernia in infants and children: In this category I feel strongly that operation except in the case of incarceration, should always involve exploration of both sides. This conclusion is an outgrowth of two experiences in which operation on the obvious side was followed in a few months by the appearance of a well developed hernia on the opposite side.

In connection with bilateral exploration I would like to point out the distinct advantage of using a curved suprapubic (Pfannenstiel type) incision. The incision provides adequate exposure for hernia repair in children and has the advantage of avoiding the fat and vascularity so annoying in the classical inguinal incision.

3. Approach in femoral hernia: I much prefer the inguinal route. While its special advantage is to be found in cases in which strangulation has occurred, I have come to use it as a routine procedure and firmly believe that in this approach one has a much better exposure of the anatomy of the inguino-femoral region.

4. Sliding Hernia: Here I must admit that I could easily "go overboard" in my enthusiasm for a particular technical maneuver. In the field of hernia surgery I

know of no change in technique that has proven as gratifying to me as has the adoption of a combined inguinal and abdominal approach in dealing with this particular problem. The simple expedient of entering the abdomen through a separate or extended incision above the internal ring has changed what had always been a bewildering and frustrating type of dissection into one that is now simple, easy and completely satisfying. By entering the free peritoneal cavity, delivering the sigmoid or caecum to a level where the whole anatomic mechanism responsible for the sliding hernia can be readily visualized, the steps necessary for correction of it become immediately apparent. By excising a wedge of peritoneum as it is reflected onto the lateral parietal peritoneum the colon can be fully reduced into the abdominal cavity and an accurate and complete closure of the sac—not just its anterior wall—can be easily effected. One has only to carry out this type of management once to be convinced that it has real merit and that it is distinctly superior to the limitations imposed by confining oneself to an inguinal dissection of sliding hernia.

The foregoing impressions and conclusions, while derived from a fairly active personal experience, are of themselves unimportant unless they have been arrived at on the basis of some degree of follow-up analysis. Only by personal post-operative examination can one fairly evaluate one's results in this field. Not only the matter of recurrence, but testicular atrophy, wound pain, paraesthesia, type of healing, etc., will often go unrecognized unless patients are specifically instructed to return for check-up examination. While I must confess that I have not made use of a well-organized tickler system, I do make a practice of periodic re-examination for at least a year in the uncomplicated case and over a period of two years in the recurrent or more involved type of hernia. In this connection I can say that this habit was largely responsible for my adoption of the McVay procedure in direct hernia. I have also learned that it is necessary to follow cases for a longer period than two years if one is to get an accurate picture of one's effectiveness in dealing with the hernia problem.

Just this week I saw a patient upon whom I had operated three years ago for a bilateral direct inguinal hernia, and who during the course of a routine physical examination was told by his physician that he had a recurrence on one side. The patient himself was not aware that he had developed a hernia. My own findings, however, confirmed the observation of his doctor.

This very recent experience, a timely one perhaps, but no less ironical, would seem to justify the title of this paper, and to emphasize what must already have been apparent, viz: that I still have much to learn about the surgical cure of inguinal hernia.

TISSUE ELECTROLYTE ABNORMALITIES
In a Case of Chronic Congestive Heart Failure

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The edema characteristically associated with congestive heart failure is believed to represent an expansion of the extracellular fluid volume, the main components of which are the electrolytes found in the extracellular space.[1] Many hypotheses have been advanced regarding the accumulation of such fluid, but irrespective of other mechanisms which may be involved, the kidney is intimately related to its pathogenesis, since in heart failure there is an inability of this organ to handle sodium, chloride, and water in a normal way. Because of these observations, the theory of forward heart failure was advanced by Merrill.[2] The urine excreted by congestive failure patients is hypotonic for sodium and chloride,[3] and diuresis induced by present day therapy increases water, sodium, and chloride excretion.[4] Thus, marked fluid and electrolyte abnormalities are encountered in patients suffering from heart failure.

The serum sodium level in patients with heart failure and edema is usually low,[5] indicating hypotonicity of the extracellular compartment. Serum potassium is usually

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normal, but some authors have demonstrated a negative potassium and nitrogen balance as the disease progresses, with a reversal of this picture as clinical improvement takes place.[6,7] Serum chloride levels may vary, and frequently a low value with a relative alkalosis may result when mercurials and resins are used in therapy.[8] Recently, the effect of digitalis therapy has been related to the potassium content of the heart muscle.[9]

The sodium retention and potassium loss are not dissimilar to the action of DOCA and other steroids,[10] and the role of the adrenal cortex in the pathogenesis of congestive heart failure is not clear, although the pattern of stress is suggested by the electrolyte findings. The decrease in filtration rate, renal blood flow, and increased filtration fraction observed in specific measurements of kidney function may be due solely to a decreased cardiac output, but these changes are not dissimilar to those found with the administration of steroid hormones.[11] Thus, in the pathogenesis of most types of congestive heart failure there are definite abnormalities in renal function, fluid and electrolyte partitions, and possibly adrenal cortical function, as well as the classical decreased cardiac output.

Since the extracellular fluid in the normal individual represents only 16% of the body weight, measurement of extracellular (serum) electrolytes gives only a sampling of the condition existing within the cell, and it must be recognized that the extracellular fluid is merely a transport medium, the vital portion of the body being the cell.

In an attempt to further elucidate the complex problems involved, a project was commenced wherein tissue electrolytes (deltoid muscle biopsy) would be measured at the same time as renal function and adrenal function, and it is the purpose of this paper to present one case of progressive cardiac failure in which detailed chemical studies were made during the course of the disease.

Materials And Methods

The patient, a 47-year-old white male, has been under treatment for hypertensive cardiovascular disease and congestive heart failure since 1950. During the past twenty months he has been under the authors' observation both as an in- and out-patient. There has been a steady progression of his illness, with increasing failure and more persistent edema, and at the present time his condition is apparently terminal.

Blood urea nitrogen, sodium, potassium, chloride, and CO_2's were done daily during the study as were analyses of 24-hour urine specimens for sodium, potassium, and chloride. Repeated urea clearances and PSP excretion tests were performed, and in an attempt to assess adrenal cortical function, several eosinophil counts following injection of ACTH (Thorn Test), and urinary 17-keto-steroid determinations, were made before and after administration of mercurial diuretics. Two muscle biopsies during hospital admissions were done during the twenty-month period of study. Sodium and potassium were estimated by means of an internal standard flame photometer,[12] and the chloride was determined by the method of Schales.[13] Standard morning urea clearances were performed, using the urease method, and ketosteroid determinations were done by the method of Drekter as modified by Keller.[14] Muscle biopsies were performed under local anesthesia, care being taken to infiltrate the anesthetic down to the muscle sheath only. On muscle, sodium and potassium estimations were performed on a wet ash preparation, chloride by the modified Volhard technic, and water content was determined by drying of the muscle to a constant weight, the fat being extracted by multiple ether washings. For comparison of data, the confidence limits of a normal series is presented, these representing the findings from ten muscle biopsies (deltoid and gastrocnemius) on five normal, healthy, white male volunteers.[15]

Results

The BUN values throughout the period of study varied from normal to slightly increased, the values being from 8 mgms.% up to a maximum of 30 mgms.% The urea clearance test remained consistently around 40% of normal, and the PSP test varied from 20 to 30% clearance in one hour. Total eosinophil counts (Thorn Test) remained consistently low, varying from 31 to 85 per cu. mm., and

the ketosteroid determination varied from 12 to 14 mgms. per 24 hours—that is within normal limits—except on one occasion, when during a severe bout of failure one reading of 5 mgms. per 24 hours was obtained, this being a low figure. The CO_2 combining power remained within normal limits until recently, when during a severe bout of fail ure low figures varying from 18-22 mEq. per liter (normal 24-34 mEq. per liter) were recorded. The electrolyte findings are summarized in the table below, which also gives the normal ranges for comparison. Urine sodium values are mentioned in the discussion.

was in an apparently terminal condition, an improvement in the extracellular electrolytes resulted in clinical improvement. This was accomplished by the administration of potassium acetate (40 mEq. daily) and several infusions of 3% sodium chloride (200 cc.) with 2 cc. of mercuhydrin and 20 cc. of aminophylline. A transient rise in renal function associated with a hypertonic salt solution along with an increased tubular mass for sodium may account for some of the changes.[22] Other newer therapeutic attempts offered this patient included the prolonged use of Dirnate (carbonic anhydrase inhibitor),[23,24] 1.4 molar diabasic sodium succinate - fumurate,[24] and

Table 1—Electrolyte Studies

		Whole Muscle (fat free)				Dry Muscle (fat free) and serum					
		L|Kg Water	mEq|Kg Sodium	mEq|Kg Potassium	mEq|Kg Chloride	mEq|Kg Sodium	mEq|Kg Serum Sodium	mEq|Kg Potassium	mEq|L Serum Potassium	mEq.|Kg Chloride	mEq|L Serum Chloride
Ranges of Normal		0.745 to 0.785	33.3 to 41.5	90.4 to 93.2	24.8 to 35.8	147 to 177	140 ±2	387 to 409	4.5 to 5.5	107 to 153	100 to 110
T. H. (M) Age 48 Congestive Cardiac Failure	Early	0.789	45	89	33	213	130	422	5.0	160	97
	Late	0.791	60	78	32	274	137	355	4.5	145	104

DISCUSSION

The low serum sodiums noted in congestive heart failure with edema may indicate a lowered osmotic pressure of the extracellular fluid, but correction of this defect is difficult and frequently impossible.[16] The explanation of this phenomenon may simply be an excessive water retention by the kidney,[17] but from the data presented, the possibility of marked intracellular migration of sodium is apparent. The low tissue potassium level is compatible with previous reports,[18,19,20,21] and the physiological implications are many. With progression of the disease in the present case, an aggravation of the existing defect was observed. By correcting for fluid content, it is seen that all of the changes may not be based on further accumulation of edema fluid and dilution of the tissue content of electrolytes only.

Urine sodium values, as would be expected, did rise with the clearance of edema fluid and would then fall again (5-20 mEq. per liter of sodium, 50 mEq. per liter of potassium. 10-20 mEq. per liter of chloride) when therapy was stopped or the patient became refractory. It is of note that when mercurial fastness occurred and the patient with these devices along with the available electrolyte data, this patient has been maintained for about twenty months. The Thorn tests did not suggest any impairment of adrenal function and the 17-ketosteroids and urea clearances did not display any specific abnormalities that could not be related to a decreased cardiac output.

SUMMARY

A case of progressive cardiac failure is presented in which detailed electrolyte studies and attempted estimates of adrenal function were made. Marked electrolytic disturbances occurred, but these could not be related to any definite impairment of adrenal function.

ACKNOWLEDGEMENT

These studies have been possible by a grant from the Delaware Heart Association.

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TREATMENT COMPLICATIONS IN A CASE OF LYMPHATIC LEUKEMIA
With Secondary Hemolytic Anemia

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Hemotological complications in patients with leukemia have received increasing attention over the past several years. One of the more common complications is acquired hemolytic anemia which can now frequently be recognized early by using the Coomb's Test to detect autoagglutinating antibodies in the serum and on the red cells. Some of these patients are able to have a lengthened span of useful life thanks to the steroid hormones.

The case to be presented is of interest because the diagnosis was difficult to establish, the treatment produced an excellent clinical response, and the terminal course was most unusual.

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CASE REPORT

A 49 year old white male was first seen in The Memorial Hospital, Wilmington, Delaware in July, 1950. At that time his blood count showed 12 gm. of hemoglobin with 75,000 white cells. The differential count was 95% lymphocytes with 5% neutrophils. The platelet count was 120,000. A diagnosis of lymphatic leukemia was made by his physician. Cervical nodes were first noted in February, 1950. No specific treatment was ordered until June of 1950 when one pint of blood was given for increased weakness. In spite of the transfusion the patient's weakness suddenly became more marked in the last week in June, and he was hospitalized.

His past history and family history were not remarkable.

System Review was significant as follows: There was no history of bruising or bleeding. Removal of all teeth four months previously had not caused excessive bleeding. There was slight dyspnea with the present illness. The urine had been somewhat dark for a few days. Stools had been black ever since iron was started several months earlier.

Physical examination showed normal vital signs with a Blood Pressure of 140/80. The patient was well-developed and well-nourished but looked quite weak. He was slightly icteric. His mucous membranes were pale. There were nodes up to 1.5 cm. in the posterior cervical chain, especially on the left. There were also several small submandibular nodes. The lungs were clear to percussion and auscultation. The heart sounds were slapping in nature, but no murmurs were heard. The liver was three fingers below the costal margin. The spleen was three fingers below the level of the umbilicus, and extended to the mid-line. Nodes up to 3 to 4 cm. in diameter were noted in the femoral and inguinal area.

The blood count on admission was 500,000 red cells with 2 gm. of hemoglobin, 69,000 white cells, and 30,000 platelets. On July 7th after transfusions, the hemoglobin rose to 11 gm. and the patient was discharged from the hospital. By July 19th the

hemoglobin had dropped to 4.8 gm. with white blood count of 15,000 and platelets of 60,000, and he was re-admitted to the hospital. The reticulocyte count was then noted to be 9.8% and the bilirubin, chiefly of the indirect type, was 5.4 mgm%. On admission his physician thought that the sudden drop in hemoglobin could be attributed to gastro-intestinal bleeding because of the history of black stools. However, the repeated drop in red cells while the patient was under close observation, plus his bilirubinemia and elevated reticulocyte count pointed to an acute hemolytic process as an explanation for the anemia. His iron therapy, as a cause of the black stools, was initially overlooked. Although most of the laboratory and clinical evidence was striking, the negative Coomb's Test was disappointing. Since 20 pints of blood were needed up to July 27th, with no obvious source of blood loss, the evidence seemed sufficient to treat the patient as an acquired hemolytic anemia. A bone marrow biopsy on July 27th showed hypercellular marrow with a considerable proportion of lymphoblasts. There were few erythropoietic forms and those seen were chiefly normoblasts. Megakaryocytes were almost completely absent.

At that time (July, 1950) adrenocorticotrophic hormones were not yet generally available. Therefore, the patient was first treated with nitrogen mustard because of its known effect on antigen-antibody relationships and on leukemia, although it was not considered the ideal drug for the leukemia itself. On July 29, 1950, after his hemoglobin had dropped to 2 gm. with a white blood count of 96,000 in spite of repeated transfusions, he was given one dose of nitrogen mustard (.2 mgm. per kg). Blood transfusions were continued. His response was good. His hemoglobin remained at 8.5 to 7 gm. for two weeks and then dropped again on August 23rd to 4.5 gm. His white count also dropped after nitrogen mustard to a low of 6,000. His reticulocyte count remained up with a high of 11% on August 14th. His bilirubin had dropped to 2.7 mgm% on August 14th but by the 23rd it rose again to 3.7 mgm%. He had deteriorated clinically in association with the blood changes. Therefore, more nitrogen mustard was given on August 23rd in the same dose as before. On August 31st the hemoglobin was 7 gm. and the white blood count was 11,500 with 13% neutrophils and 85% lymphocytes. The patient improved clinically, but it soon became obvious that the nitrogen mustard was only transiently affecting his hemolytic process. From July 1st until the time of the first (July 29th) nitrogen mustard, the patient had received 28 transfusions and from the first mustard until August 31st he needed ten more.

On September 6th, when he had 5.5 gm. of hemoglobin and a white blood count of 6,900 with 20,000 platelets, x-ray therapy was started to the spleen with 50 r. at 70 cm. through a 14 x 14 port for two doses. This seemed to afford him additional relief and it reduced the size of the spleen. However, by September 27th the hemoglobin was found to be 4 gm. with a red blood count of 100,000; white blood count 5,900 and platelets of 20,000. The reticulocyte count was then .3%. The bilirubin remained at 2.5 mgm%. The bone marrow was reported only as compatible with chronic lymphatic leukemia.

On September 27th ACTH-Wilson was obtained and started on 10 mgm. q.6 hrs. This was increased to 15 mgm. q.4 hrs. on October 2nd because of a limited eosinophile response (from 274 to 200). By October 6th the hemoglobin had risen to 5.5 gm. and the white blood count to 21,000 without transfusions, and the patient felt much better. Up to this point from July 1st he had been given 42 pints of blood. His response to ACTH was therefore counted as further evidence in favor of hemolytic anemia in spite of the persistently negative Coomb's Test. On October 7th the dosage of ACTH-Wilson was dropped to 15 mgm. q.8 hrs. By October 9th the hemoglobin was 8 gm.; red blood count 1.8; white blood count 21,000; reticulocyte count 5%; and the spleen had become one-half its original size. It had shown only a slight response following the x-ray therapy prior to the ACTH, but a delayed response to x-ray could not be ruled out. In the middle of October, 1950, the ACTH supply ran out for a few days. The spleen increased slightly in size but the count re-

mained at 9 gm. of hemoglobin and 8,000 white blood count. The dose of ACTH-Wilson was dropped to 10 mgm. q.8 hrs. At that time the patient felt as well as he had been in the spring of 1950. His bilirubin was then 1.2 mgm. He was continued on ACTH with improvement except for a slight drop in his hemoglobin from 10 gm. to 9.5 gm. and slight change in the size and consistency of the spleen. After the first of November the supply of ACTH was again interrupted for 2 to 3 days. Later, the dosage of ACTH was reduced to 10 mgm. q.12 hrs., but the hemoglobin dropped slowly to 8 gm. by November 10th. The dose was then jumped to 40 mgm. per day. The hemoglobin did not go up until November 21st when it reached 9.5 gm. By then the patient was able to return to full time work On December 1st the spleen was irradiated again since it had shown no change during the preceding two months. It was given 100 r. in air through a 20 x 12 port at 70 cm. with a 200 KV for a total of 3 anterior ports and 2 posterior ports or an estimated tumor dose of 240 r. Although there had been little change in the spleen or in the blood count, on December 8th it was decided to try stopping the ACTH. The count on that date showed a hemoglobin of 10.5 gm. and white blood count of 10,000 with 12% seg. and 88% lymphocytes. The reticulocyte count was 9%. The patient did fairly well for about a month, although in the interim the spleen had increased in size in spite of the x-ray therapy. On January 2, 1951, he lacked his usual sparkle and pep, and the hemoglobin was found to be 9 gm. On January 5th, because of a further drop in his hemoglobin to 8.5 gm. with a white blood count of 5,700, reticulocyte count of 1.3%, and bilirubin 1.04 mgm, cortisone was started at 100 mgm. daily I.M. In four or five days the spleen shrank from 13 cm. below the costal margin to 10 cm. from the costal margin. The patient was clinically improved with a hemoglobin rise to 10 gm. and a red blood count jump from 2.34 to 3.07. The white blood count was 10,000. By February, 1951, he was on oral cortisone 25 mgm. t.i.d. The spleen was only 9 cm. from the costal margin and the hemoglobin was up to 12.5 gm. The dose of cortisone was then dropped to 60 mgm. per day and was continued at the same rate to May, 1951, when it was reduced to 25 mgm. b.i.d.

During all this period of steroid therapy there had been relatively few side effects. The patient worked full time on a laundry truck without difficulty. He had been on a restricted sodium diet and on 2 to 3 gm. of KCl per day. The blood pressure varied between 120/70 and 140/90. The blood sugar (feasting) reached 190 on one occasion but no glycosuria was noted. His weight gradually rose from 150 to 165 but there was no overt sign of edema. The weight gain was thought to be real weight consistent with his improved appetite. Mild axillary adenopathy with several nodes up to 1 to 2 cm. in size remained relatively constant. There were no intercurrent infections until June, 1951, when he developed bronchitis which responded to penicillin.

By August 17, 1951 his hemoglobin was 12.5 gm. and white blood count 8,000 with 36% seg; 2% non-seg. 56% lymphocytes; 1% monocytes and 5% esoinophils. The spleen remained at 9 to 10 cm. below the costal margin. By October 2nd he had done so well that it was thought safe to reduce the cortisone to 25 mgm. per day. On October 17th his hemoglobin dropped to 11 gm. and he had another episode of acute bronchitis, once again easily controlled by penicillin. A drop of the hemoglobin to 10 gm. by October 30th was thought to be sufficient reason to raise the cortisone to 50 mgm. per day and on November 2, 1951, further x-ray treatment of the spleen was started through an 18 x 16 port for 100 r. in air times four. By November 23rd, the hemoglobin had risen to 13.5 gm. and the white blood count to 11,400. There was a slight increase in the size of the spleen and it was thought permissible to drop the cortisone again to 25 mgm. per day. The patient did well through December, 1951. On December 31st, the hemoglobin was 12 gm. with a white blood count of 8,300.

On January 14, 1952, purpura was noted on the legs below the knees, with areas 2 to 4 mm. in size. The platelet count then was 64,000, but by January 18th it had dropped

to 34,000. The spleen remained about the same. The patient was again hospitalized for study. Platelet counts varied from 30,000 to 60,000. Clot retraction was normal; the hemoglobin and white blood count showed no change. The bone marrow was still that of a chronic lymphatic leukemia. Megakaryocytes were present. The cortisone was increased to 200 mgm. per day. On the increased cortisone, the spleen shrank slightly more in size, and the patient showed subjective improvement, although there was little change in the platelet count. The purpura faded on bed rest but on February 4. 1952, after he had been allowed home for a few days, the purpura became worse especially on the legs. In addition, a few petechiae were noted on the hard palate and over the trunk. The platelet count was 20,000, white blood count 19,000, and hemoglobin 13 gm.

The patient was re-admitted for a splenectomy on February 7th. His immediate response was good. There was a rapid rise in the platelets to 48,000 by February 9th and to 180,000 by February 24th. The white blood count which had averaged around 20,000 also rose to 35,000 to 40,000 The spleen pathologically showed chronic lymphatic leukemia plus one 4 to 5 cm. area of infarction. It weighed 1680 gms. On February 19th, after the patient had been up and about and had started to eat, signs of intestinal obstruction developed and necessitated a re-operation on February 24th. Enlarged mesenteric lymph nodes were found which had not been noted at the time of the first operation. There were also adhesions binding down and apparently obstructing part of the colon. These were not where the surgeon would have expected them if they had been caused by the splenectomy. Although the adhesions were freed, the patient did not do well post-operatively. He developed recurrent abdominal pain and signs of intermittent small bowel obstruction. This was thought possibly to be related to enlarged mesentric lymph nodes. Cortisone 100 mgm, had been given daily up to February 14th when it was dropped to 50 mgm. per day. His general condition was poor. As a desperate measure x-ray therapy was started to the root of the mesentery in the abdomen but the patient's course went rapidly down-hill. There was only time for one x-ray treatment . . . he died on March 2, 1952.

POST-MORTEM EXAMINATION

Post-mortem examination revealed the following significant findings: There was some congestion in both lung bases. Clotted blood was present in the anterior descending coronary artery. The liver weighed 2380 gm. Ninety cm. of the ileum was swollen and dark red in color; and the walls were engorged with blood. Multiple thrombi were present in the mesenteric vessels going to this section of the bowel. The lymph nodes were enlarged and indurated throughout the body. On microscopic examination, the liver showed scattered infiltration of round cells in the lymphoid series, some of which were immature. The adrenals seemed normal although their natural yellow color was absent. There was some infiltration of the kidney capsule by leukemic cells. The usual structure of the lymph nodes was replaced by a diffuse infiltration of rounded cells of the lymphocytic series, many of which were immature.

DISCUSSION

This case illustrated many of the problems in the diagnosis and treatment of hematological complications of leukemia. The patient had an excellent response to steroid therapy but an unexpected end result. His sudden onset of anemia was initially ascribed to gastro-intestinal bleeding. It was not until his second hospital admission in the middle of July 1950 that a hemolytic process was suspected. The Coomb's test was disappointing, but the negative result was perhaps more of a reflection on the way the test was done than on the diagnosis although the presence of hemolytic anemia with a negative Coomb's test has been mentioned in the literature.[1,3] The high reticulocyte count, increased indirect bilirubin and need for large amounts of blood without obvious blood loss seemed to be sufficient evidence for the diagnosis. The patient's response to therapy and his clinical course strengthened the impression of acquired hemolytic anemia.

Determination of fecal urobilinogen would have been of confirmatory value.[4]

The initial bone marrow report, which noted a crowding out of the erythroid forms, suggests that the anemia also had "myelophthisic" features. Since some studies have shown that many leukemics have red cells with shorter lives than average, it should be remembered that this mechanism can also of itself eventually lead to severe anemia in leukemia without the more dramatic picture seen in an acute hemolytic process.[5] This patient's anemia was most likely due to a combination of these factors. The treatments, all of which had some primary effect on the underlying leukemia,[4,2,6] are therefore hard to evaluate in respect to the hemolytic process alone.

From a theoretical point of view an abnormal antigen antibody relationship is thought to form at least one of the mechanisms for the hemolytic reaction.[9, 8,1] Nitrogen mustard which was given to the patient for this reason, probably did also have some effect on the leukemia itself as can be gauged by the drop in the white count. One cannot prove, but can only speculate, whether the beneficial effect on the anemia in this patient was because of the clearing out of the overcrowded leukemic marrow or because of the interruption of the hemolytic process. The fact that the reaction only lasted about two weeks seems to be in favor of the viewpoint that the nitrogen mustard acted as a desensitizing agent. In other conditions where nitrogen mustard has been used for its blocking effect on antigen antibody reactions, its effective duration is about two weeks.[10]

Radiotherapy to the spleen for secondary hemolytic anemia is not usually recommended. However, it is often used to control the level of the white cell count. This patient did seem to show clinical improvement after the initial use of radiotherapy in September 1950. The exact reason for the improvement is not clear, but the relatively small effect on the white blood count in relation to the shrinkage of the spleen and the improvement in the hemoglobin certainly suggest that splenic irradiation may have helped interrupt the hemolytic process. This has subsequently been demonstrated in another case where no other therapy was used.

Adrenocorticotrophic hormone was not used sooner in this patient because it was not available. Furthermore, its use for such cases had not been generally stressed early in 1950. Most of the reports on its success in the treatment of hemolytic anemia have been subsequently published. ([11, 12, 13, 14, 15, 16]) One to two-year end results have seldom been mentioned. This patient's response to ACTH was much more dramatic than to any of the therapy preceding it. No blood was needed from then until terminally. In December, 1950, radiotherapy was used with the hope that the combined effect of ACTH and x-ray might be better than either alone. The fact that there was some brief stabilization of the hematological status indicated that x-ray may have played a small part in the response since in November there had been a slight but rapid drop in hemoglobin when ACTH was stopped for only a few days at a time. The third time x-ray was used there was no definite response.

The switch to cortisone was effected without difficulty and in fact the results seemed somewhat better than with an equivalent dose of ACTH. This too cannot be made as an unequivocal statement since variation in the natural course of the disease may well have been more responsible than the treatment. The use of cortisone for a year without complications is perhaps worthy of note.

The development of purpura was an unexpected complication but it has been described in leukemia as part of the "hypersplenism" picture.[17] While the purpura seemed to improve somewhat to an increased dose of cortisone, the response was transient. It is known that the response of thrombocytopenic purpura to steroid therapy may not be constant or permanent.[18] It nevertheless is of interest that the purpura developed while the patient was on cortisone. It was with some trepidation that splenectomy was finally advised, but it seemed the only "out,"[19] aside from doing nothing, and the patient was still a good operative risk. The immediate response to surgery was more than had been expected. With the

good platelet response, it was hoped that there might also be a beneficial effect on the hemolytic anemia since it too can be treated by splenectomy.[9, 16, 20, 21]

The cause of the final flare-up of the mesenteric lymph nodes and the development of mesenteric thrombosis is unknown. The leukemia itself, loss of the spleen, the surgery, and the cortisone all may have played a part. The relative importance of each is unknown. We can only report the terminal events and hope that explanations will be forthcoming as our understanding of leukemia is improved.

SUMMARY

A case of chronic lymphatic leukemia with secondary hemolytic anemia was reported because of the various types of therapy used, including blood, nitrogen mustard, irradiation of the spleen, ACTH, cortisone, and finally splenectomy. The response to ACTH and cortisone was dramatic and was maintained for 18 months, following which thrombocytopenia with purpura developed although there was no diminution in effect of the steroids on the hemolytic process. The splenectomy was successful surgically and hematologically, but the patient died four weeks postoperatively from mesenteric thrombosis and a flare of his leukemia as manifested by rapid enlargement of the mesenteric lymph nodes in his final month of life.

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BREAST LESIONS

A Review and Classification

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This study is based on the breast lesions encountered at The Memorial Hospital during the five year period 1945 to 1949 inclusive. The material embraces a total of 375 distinct lesions. Diagnosis in each case is based on histological study.

The lesions of the breast are many and varied, and no universally accepted classification has been proposed at the present time. Confusion and ambiguity exist in the nomenclature, both in the etiology and in the cellular origin of many of the lesions.

A major factor in this diversity of opinion is the constantly changing status of mammary tissue. There is a wide fluctuation in the functional activity and in the morphology of the normal breast, which is subject to physiological hypertrophy and involution; changes which affect both the parenchyma and the stroma. In disease, these changes may become accentuated and erratic. These variations may involve the breast in whole or in part, and more than one change may be present in the same breast. Many lesions, such as lobular hyperplasia and fibro-adenoma, show evidence of perversion or normal physiological changes to a marked degree, while neoplasms are profoundly altered in their behavior and morphology by these factors, of which the effect of pregnancy on mammary carcinoma is a striking illustration. An ap-

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preciation of these factors helps to explain the almost infinite variation that breast lesions may assume.

Of the lesions in this series there are 108 benign neoplasms. These are considered under the headings of Duct Papilloma and Fibro-adenoma.

Duct Papilloma

There is a total of 22 breast lesions in the series which are classified as duct papillomas. A variety of names are used to indicate these lesions and include adenoma, duct papilloma, proliferating cystadenoma, adenocytoma, intracanalicular cystadenoma, papillary cystoma, villous papilloma, and intracystic papilloma. These tumors are usually solitary, but in an appreciable number of cases may be multiple. Characteristically, the lesion is located in the larger ducts under the nipple and in the central zone of the breast, the peak incidence occurring between forty and forty-five years of age, but they have occurred shortly after puberty as well as long after the menopause. Parity seems to have no influence on the incidence.

The tumors vary in size from microscopic lesions up to several centimeters in diameter. They are encapsulated growths that contain serous or sanguinous fluid which bathes the epithelial tufts, this explaining the frequency of bleeding from the nipple as a leading symptom in the disease. The tense, unopened cyst, is often of a bluish or purplish hue, and its cavity may be partially or wholly filled by the papilloma. Quite often the tumors appear in dilated, tortuous ducts rather than in typical cystic structures. The papilloma is usually attached to the cyst wall at its base only, and a delicate, branching connective tissue framework transmits blood vessels to the epithelial covering.

Microscopically, the arborescent, fibrous stalks usually exceed the epithelial elements in amount. The epithelium is one or two layers in thickness, orderly in arrangement, and the outer layer is composed of tall columnar cells with a secretory border. The nuclei are regular in size and staining reaction, and mitotic figures are scarce.

Frequently, however, the benign papilloma presents a more active picture, and proliferation, with frequent mitotic figures, may be marked. The point at which a benign papilloma approaches malignancy is often not sharply defined. Two lesions in the present series are placed in the benign category with some trepidation. The usual criteria of malignancy are: invasion of tissue through the basement membrane by epithelial growth, secondary attachment of the papilloma to the cyst wall, minimal or absent stroma, and areas of necrosis. Many benign papillomas ultimately undergo malignant transformation. This propensity is reflected in the attitude of some pathologists who classify this lesion as a grade I adenocarcinoma.

Fibro-Adenoma

Eighty-six lesions in the series are classified under the general heading of fibro-adenoma. Included in these tumors are 42 pericanalicular fibro-adenomas and 36 intracanalicular fibro-adenomas; 6 lesions are classified simply as fibromas. There are 2 cystosarcomas phyllodes in the series. Eight lesions of the male breast known as gynecomastia are also included.

The pericanalicular and intracanalicular fibro-adenomas are circumscribed fibro-epithelial overgrowths which are usually limited by a distinct capsule. Some fibro-adenomas are difficult to distinguish from certain types of lobular hyperplasia. The peak incidence of these tumors is in the age group 20 to 25 years. Occasionally they are found shortly after the onset of puberty and following the climacteric, parity seems to have little effect on the incidence, and etiologically, estrogenic stimulation is an important factor in their development.

Multiple pericanalicular and intracanalicular fibro-adenomas do occur, but the lesion is usually solitary. They may appear in any part of the breast, but are more frequent in the upper outer quadrant. The maximum size attained by these tumors is 5 to 6 cm., and many remain stationary for long periods after reaching a size of 2 to 3 cm. The tumor is responsive to physiological stimuli in the same manner as normal breast tissue, thus enlargement of the tumor may be rapid in pregnancy. Malignant change is a most unusual happening.

Macroscopically, fibro-adenomas are seen to be sharply circumscribed growths which are

easily separated from the surrounding breast tissue. They are firm, pinkish or grayish-white, spherical or oval tumors. On section the cut surface is somewhat convex, slightly granular, and homogeneous. Lobules and papillary excrescences may be noted in the intracanalicular variety, and cystic spaces may be present.

Microscopically, the pericanalicular fibro-adenoma appears as an exaggeration of the hypertrophy seen in the adolescent breast. The acini and ducts are more numerous and are surrounded by a stroma of fibrous tissue. Reduplication of the epithelium is often present, and an occasional mitotic figure may be seen. The stroma is formed by proliferation of the normal perilobular and periductal connective tissues, the interlobular connective tissues taking no part in the process. The stroma shows much variation in density, and the capsule is formed by a definite sheath of dense fibrous tissue.

The intracanalicular fibro-adenoma presents a distinctive appearance microscopically. There is seen a remarkable proliferation of connective tissues which projects into the ducts in the form of polypoid masses. A dilated duct often appears to be crowded with masses of fibrous tissue, each covered by a layer of epithelium invaginated from the duct wall. The section often gives the appearance of branching slits lined by epithelium. The connective tissue stroma of the intracanalicular fibro-adenoma is usually very loose in texture and often has a myxomatous appearance; hence these tumors are often referred to as myxomas. It may be noted here that frequently gradations are soon between the pure pericanalicular and the pure intracanalicular fibro-adenoma. Both types of proliferation may be seen in the same tumor.

The cystosarcoma phyllodes or giant intracanalicular fibro-adenoma is a variant of the intracanalicular fibro-adenoma which is usually found near the time of the menopause. These tumors are slow growing and may reach an immense size. On section, cystic spaces are found into which project fibrous polypoid masses. Microscopically, myxomatous connective tissue predominates. The polypoid fibrous structures are covered by layers of columnar or cuboidal connective tissue.

Cystosarcoma phyllodes is usually considered to be a benign lesion. However, the connective tissue frequently shows areas of increased cellularity wherein are seen pleomorphism, frequent mitotic figures, and variation in staining reactions. Here a sharp differentiation between a benign lesion and a true sarcoma may be extremely difficult.

For the present purpose a fibroma of the breast is considered to be a form of fibro-adenoma in which the epithelial elements have been crowded out and replaced by connective tissue.

GYNECOMASTIA

In the series of breast lesions are 8 lesions of the male breast which are classified as gynecomastia or fibro-adenoma of the male breast.

In this lesion there is a nodular enlargement of the male breast, and microscopically, there is a growth of the mammary ducts and the periductal stroma. The epithelium is hyperplastic and papillary projections may form. A periductal infiltration of chronic inflammatory cells may be present, including lymphocytes, plasma cells, large mononuclear cells, with occasional eosinophile and polymorphonuclear cells.

These lesions appear to be dependent on a hormonal imbalance, and may be associated with testicular teratomas or hypophyseal adenomas. Trauma is often a factor in accelerating the rate of growth in the breast, but there is doubt that it is a primary cause.

LOBULAR HYPERPLASIA

Of the remaining benign lesions, 64 are classified as lobular hyperplasia. Fifty-five of these are of the non-cystic type, eight are of the cystic type, and one case is classified as sclerosing adenosis.

The term lobular hyperplasia, first proposed by Smith[1] in 1940, with its divisions of cystic and non-cystic, both of which may be localized or generalized, embraces all of those conditions formerly referred to by a host of terms among which are chronic mastitis, chronic interstitial mastitis, cystic disease of the breast, chronic cystic mastitis, diffuse fibro-adenoma, cystadenoma papilliferum, involution cysts, abnormal involution of the breast, adenosis, Schimmelbusch's disease, and many others.

There is much disagreement as to the true nature of this disease. It is regarded by some as inflammatory because of the frequent lymphocytic infiltration. Others regard it as a neoplastic disease. Still other observers think of it as a perversion of involution. The basis of the inflammatory theory is the lymphocytic infiltration, a finding that is always present in the involution following lactation and which is also often seen in senile involution. It is difficult to consider the disease as a neoplasm as there is no true tumor. Epithelial hyperplasia may, at times, suggest a neoplastic process, but a similar type of hyperplasia occurs in benign hyperplasia as seen in the prostate and in the thyroid gland.

Breast tissue, parenchyma and stroma, is greatly influenced by external stimuli, and these factors may result in hyperplasia which is succeeded by involution. Normally an equilibrium is maintained between the various components of breast tissue. In lobular hyperplasia all of the changes that occur in a normal breast are seen, but the normal proportions are lost, and hyperplasia may affect the parenchyma, the stroma, or both together in varying proportions, while succeeding involutional changes may likewise proceed in an erratic manner. The changes seen in lobular hyperplasia, including the epithelial hyperplasia and atrophy, cyst formation, and fibrous overgrowth, are best explained on this basis. The consensus of modern opinion appears to favor this view.

Grossly the lesions of lobular hyperplasia vary widely. The tissue is firm and rubbery in consistency, yellowish-white in color, and is not encapsulated or shaply circumscribed. The cut surface often reveals raised pink and gray dots of newly-formed glandular tissue, dilated ducts often containing a putty-like material that may be expressed as a cast of the duct. Cysts of varying size and number are often seen, which when unopened are bluish in color due to reflection of bluish light from the tense cyst wall, the color disappearing after rupture. In the non-cystic form of lobular hyperplasia minute cysts may be present, and it is usual to apply the term cystic to those cases in which cysts of 1 cm. or more in diameter are present. The cut surface presents an ill-defined area rather than a lump as seen in carcinoma, and the stony hardness, the grittiness to the knife, and the yellow streaks and specks characteristic of infiltrating carcinoma of the breast are not seen.

There is also a great variation in the microscopic picture in lobular hyperplasia. The epithelium in the breast ducts forms multiple layers and gives rise to papillary processes. Epithelial budding and spilling may mimic the invasiveness of infiltrating carcinoma. Atrophy of the epithelium may be a feature, the cells lining an acinus may become more and more indistinct, the acinus finally becoming replaced by fibrous tissue. Persistent epithelium may be cuboidal or columnar and contain an increased amount of eosin-staining cytoplasm, this type is often seen lining the cystic spaces. The smaller ducts and acini may assume the appearance of sweat glands. Round cell infiltration about the ducts and acini is frequently seen.

Selerosing adenosis or adenomatosis, also known as fibrosing adenosis, is a relatively uncommon variant of lobular hyperplasia. The condition is usually seen in the central zone of the breast in association with the larger ducts, and grossly the involved area appears as a dense, hard, often multiple lesion that may have practically all of the characteristics of an infiltrating carcinoma. The ordinary variety of lobular hyperplasia is present in the adjoining breast tissue. Microscopically, the lesion may closely resemble carcinoma, since a dense, fibrous, connective tissue surrounds solid isleands and cords of glandular epithelium, about which a basement membrane is absent or ill-defined, the cells being compressed by the heavy fibrous overgrowth. Scattered throughout the lesion are acinar structures of varying size. Careful study reveals that the cords and strands of epithelium are epithelial buds that have been pinched off by the proliferating connective tissue, and the epithelial cells, showing no mitotic figures, are uniform and of medium size.

Of great practical importance is the differentiation of lobular hyperplasia from mammary carcinoma. The intraductal type of carcinoma frequently shows areas of central necrosis, a finding not seen in lobular hyper-

plasia, and minute papillomas are unusual in carcinoma but are common in lobular hyperplasia. In lobular hyperplasia the cells are benign in appearance, mitoses are infrequent, and the cells are more uniform, in marked contrast to carcinoma in which mitotic figures occur frequently and pleomorphism is usually marked.

It must be stressed that the differentiation of lobular hyperplasia from mammary carcinoma cannot be made from one feature alone; only by a summation of all the findings, gross and microscopic, can the true diagnosis be established.

FAT NECROSIS

Three of the lesions in the series are classified as fat necrosis. Mammary fat necrosis is a condition that is frequently preceded by trauma, but in many cases no such history can be obtained. Fat necrosis grossly forms a small, dense nodule that often resesmbles a carcinoma, and the appearance is enhanced if cholesterol and calcareous material accumulates in the lesion. The lesion consists of a chronic granulomatous type of nodule which is stellate or lobulated in shape with surrounding fibrosis. Microscopically, the condition is characterized by fibrous tissue, lymphocytes, and foreign body giant cells, together with foam cells which are lipoid macrophages. Spaces formed by cholesterol crystals are often seen in the mounted section, and areas of calcification are not uncommon. The parenchyma of the breast is not involved in the process.

PLASMA CELL MASTITIS

This lesion is rare and only one example occurs in this series. The condition has a rapid onset with marked tenderness and a puriform discharge from the nipple, and resolution is very slow, an indurated nodule persisting indefinitely. The lesion is a periductal mastitis arising in association with dilated ducts beneath the nipple, probably a chemical reaction due to retained secretion. Clinically, the lesion has many of the characteristics of mammary carcinoma. Examination of the gross specimen reveals a mass of radiating, dense, fibrous tissue strands with dilated ducts containing creamy, puriform material, and microscopically, the lesion is one of subacute inflammation with plasma cells predominating, the preponderance of plasma cells being characteristic. Epithelioid cells and foam cells are commonly present and may stimulate tuberculosis.

TUBERCULOSIS

Included in the series is one tuberculosis lesion. Tuberculosis breast lesions are rare, the infection entering the breast by retrograde spread through the lymphatics or by direct spread from tuberculosis osteomyelitis of the underlying ribs. It is usually secondary to tuberculosis cervical adenitis or to pulmonary tuberculosis.

Grossly the lesion consists of a chronic induration of the breast tissue which has a tedency to break down with sinus formation.

Microscopically, the lesion is characteristic. This consists of central caseation with surrounding epithelioid cells, giant cells of Langhan's type, and lymphocytes, surrounding which is a layer of fibrous tissue. The lesions are usually multiple.

MALIGNANT LESIONS

Of the 375 breast lesions in the series, 189 are classified as malignant tumors, 188 of these are carcinomas, while there is only one sarcoma.

CARCINOMA

Included in the 188 breast carcinomas are 166 lesions diagnosed by tissue sections and 22 by aspiration biopsies of the breast.

The 166 lesions in which tissue sections are available are herewith divided into two categories; viz., extraductal carcinoma and intraductal carcinoma. A total of 131 lesions are classified as extraductal, and 35 as intraductal.

The extraductal lesions are made up of the following subtypes:

1. Infiltrating, spehroidal cell carcinoma	109	
2. Infiltrating adenocarcinoma	21	
3. Medullary carcinoma	1	
	131	131

The intraductal carcinomas are divided into the following subtypes:

1. Comedo carcinoma	32	
2. Papillary adenocarcinoma	1	
3. Mucoid carcinoma	2	
	35	35
		166

This classification is based on the predominant feature in the different tumors, but it must be appreciated that there is no hard and fast boundary line between the various types of mammary carcinoma. The broad divisions of extraductal and intraductal are somewhat arbitrary. The intraductal growth may break out of the ducts and assume all of the characteristics of an extraductal tumor. However, this division has some justification in the gross and microscopic appearances as well as in clinical behavior A carcinoma that is intraductal tends to follow a less malignant and slower course, metastases occurring much earlier in the tumor that is extraductal from the beginning. Frequently a predominately undifferentiated spheroidal cell carcinoma may be fairly well differentiated at some points and approach the highly cellular medullary type in others. It will thus be realized that there is no absolute, exact classification possible and that there is considerable overlapping of the different types.

Infiltrative Spheroidal Cell Carcinoma

The commonest occurring carcinoma of the breast is the infiltrative spheroidal cell carcinoma. This lesion has been variously designated as carcinoma simplex, lobular carcinoma, and scirrhous carcinoma. This neoplasm metastasizes early by lymphatic spread to the axillary and clavicular lymph nodes. In the present series axillary nodes were involved in 58 casse, representing slightly more than 60% of the total 96 lesions in this subgroup.

Grossly, infiltrative spheroidal cell carcinoma consists of a tumor that is firmly adherent to the surrounding tissues, sending radiating processes out into the fat, appearing densely hard to palpation, and when incised, cuts with a gritty, calcareous sensation, not unlike that experienced in cutting an unripe pear. The cut surface is concave due to retraction of the fibrous tissue, grayish in color, and pale yellow dots and specks may be seen.

Microscopically, the normal breast tissue is seen to be replaced by small masses, strands, and columns of malignant cells, often compressed by fibrous tissue into polygonal and distorted shapes. Lymphocytic infiltration is frequent, and mitotic figures may be infrequent. The proportion of fibrous tissues varies greatly but is usually very dense and abundant. Invasion of lymphatics and veins is frequently seen.

Infiltrative Adenocarcinoma

This lesion is usually considered to be of a lower degree of malignancy than the spheroidal cell carcinoma. However, this is not borne out by the present series of 21 cases in which the axillary lymph nodes are involved in 11, representing slightly more than 73%. The term is used here to denote the well differentiated carcinoma with marked glandular formation, the cells bearings a close resemblance to those of normal ductal and acinar epithelium. Mitotic figures are fairly common, and the fibrous tissue reaction approximates, but is not quite so well marked as in the spheroidal cell carcinoma. In all other details these lesions are similar.

Medullary Carcinoma

This term has been applied to several types of breast carcinoma but is here used to denote a highly cellular, rapidly growing tumor with little or no fibrous tissue reaction. The tumor is soft, spreads rapidly, and metastasizes to the axillary lymph nodes, and ulceration of the skin occurs early. The course of the disease is comparatively rapid.

Microscopically, masses of large, round, pale epithelial cells are separated by a scanty and delicate stroma. Mitotic figures are numerous, and degeneration and necrosis are common in such tumors.

Two lesions in the present series are herewith classified as medullary carcinoma.

Intraductal Carcinoma

A total of 35 mammary lesions are placed under this heading. These are composed of 32 comedo carcinomas, 1 papillary carcinoma, and 2 mucoid carcinomas. As a group intraductal carcinomas are much slower in their rate of growth than the extraductal growths. Often they remain circumscribed by the mammary tubules for long periods before breaking out into the stroma of the breast. This characteristic contrasts with the extraductal carcinomas which invade the breast stroma from the beginning. It must be appreciated that intermediate types frequently occur.

Comedo Carcinoma

In this form of intraductal carcinoma,

plugs or casts of carcinomatous cells may be expressed from the severed ducts when the tumor is sectioned, this property giving rise to the term "comedo." These tumors are of relatively slow growth and often a number of years may elapse before the growth attains a large size. Metastases occur later, and hence this tumor offers a more favorable prognosis than in the extraductal growths. This lesion frequently occurs in a breast which contains areas of lobular hyperplasia. There are 32 comedo carcinomas in the present series. Axillary lymph node metastases are seen in only three of these cases, an incidence of less than 10%. This is in striking contrast to the incidence of lymph node metastases in the extraductal neoplasms.

Comedo carcinoma occurs most frequently in the central zone of the breast, often beneath the nipple, but it may appear in any area.

Grossly, the comedo carcinoma has not the stony hardness found in the spheroidal cell carcinoma. The ramifying, dense connective tissue is not seen, and the tumor is less sharply defined than the infiltrative carcinoma. Mobility of the tumor is late to disappear. The characteristic tumor casts of the ducts appear on the cut surface when slight pressure is applied.

Often the diagnosis between lobular hyperplasia and comedo carcinoma is not possible by gross examination and the diagnosis must be made with the microscope. Microscopical sections reveal the ducts, which vary in size and shape, to be lined by many layers of cells, and some of them are completely filled by the proliferating epithelium. The tumor cells may show no marked malignant characteristics, often containing small, dense nuclei, and a slight degree of pleomorphism with an occasional mitotic figure may be seen. A loss of polarity in the cellular arrangement is often a prominent figure, and central necrosis is common in the tumor-filled ducts. On careful search the epithelium in one or more places may be seen to invade the surrounding fat and fibrous tissue, a feature which, when present, establishes the diagnosis of malignancy beyond question. The papillary outgrowths of epithelium within the ducts usually seen in lobular hyperplasia is seldom seen.

PAPILLARY ADENOCARCINOMA

One lesion in the series is classified as a papillary adenocarcinoma.

This malignant lesion arises in a dilated cystic duct, and it often represents malignant change in a benign intraductal papilloma. The tumor usually occurs in the central zone of the breast, causing a sanguinous discharge from the nipple. Rapid growth is frequently seen with this type of growth, which may reach a diameter of 10 cm. or more. On gross examination the tumor is soft and pliable, due to the absence of fibrous tissue.

Microscopically, the epithelium is arranged in stalks, folds, and lace-like patterns. Acinar structures are seen which are similar to but larger than those in the benign papilloma, and a fibrous stroma is almost non-existent. The cells are often many layers in thickness with frequent mitotic figures and marked pleomorphism. A distinguishing feature is the attachment of the growth to the ductal margins with indications of invasion. At times the distinction between benign and malignant papillomas of the breast is a most difficult one to make. This is reflected in the attitude of a number of pathologists who consider the apparently benign papilloma to really be a malignant papilloma of the lowest grade of malignancy.

MUCOID CARCINOMA

This rather uncommon tumor is one of the slower growing carcinomas that may be present for years, and reach a large size. Metastases are late in appearing and this tumor offers a relatively good prognosis.

The term "mucoid carcinoma" is used here to designate the circumscribed form in which the gelatinous material occurs throughout the tumor, and it may be noted that mucoid degeneration may occur in the infiltrative type of mammary carcinoma. When this occurs the growth is much slower in these areas, this appears to be due largely to inadequate nutrition which is a result of the surrounding mucoid secretion. In the typical mucoid carcinoma the tumor often appears to be soft and cystic, the mucoid material being very soft and semi-fluid.

Microscopically, the epithelium has malignant characteristics and may be arranged in an acinar or papilliferous fashion. The cells contain varying amounts of secretion and signet ring forms may be seen. The gelatinous propensities are an inherent property of the malignant cells and gelatinous secretion is present in the metastases from this tumor.

Carcinomas—Unclassified

An additional 22 cases of carcinoma of the breast are diagnosed by aspiration biopsy, and tissue sections are not available. The malignant nature of the lesions is established but the type of carcinoma is undetermined. These cases represent lesions that were inoperable and cases which did not receive treatment at this hospital.

Sarcomas

There is one mammary sacoma in the series, the lesion representing sarcomatous change occurring in a benign intracanalicular fibroadenoma.

Sarcomas may arise in a pre-existent fibroadenoma or may appear without this antecedent. Any of the mesodermal elements in the breast may undergo sarcomatous change and fibrosarcomas, leiomyosarcomas, liposarcomas, etc., rarely occur. The growth is usually of the fibrous spindle cell type.

These tumors may be circumscribed or infiltrating. They are usually composed of solid, fleshy tissue which grows in interlacing bands which often enclose softer, myelomatous material.

Microscopically, the mammary sarcoma is composed of interlacing bands of fibro-spindle cells often arranged in a whorled fashion. Occasional mammary tubules are seen. The sarcoma cells show marked pleomorphism with dense, bizarre nuclei, and mitoses are numerous.

Summary

The material for this paper consists of the mammary lesions observed at The Memorial Hospital during the five year period 1945 to 1949 inclusive. A reclassification of the 375 lesions is presented based on histological examination of the microscopic sections contained in the hospital files. The diagnostic criteria of the various lesions are reviewed.

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CYSTADENOCARCINOMA OF OVARY
Twenty Years Survival After Known Peritoneal Metastases
A Case Report

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It is a well-known fact that serous and pseudomucinous tumors of the ovary are notorious for their unpredictable and often bizarre behavior. This has been substantiated so often that their vagaries are accepted by physicians who are in a position to follow such cases and note these departures. A case of papillary cystadenocarcinoma of the ovary was observed recently in the Memorial Hospital that not only exhibited an unusual site of metastatic spread but has, in addition, survived twenty years after known peritoneal implants were observed.

A. B., a 72 year old white female, was first seen in the out-patient department in October, 1952. Her complaints at that time were confined to her sternum and consisted of a dull, boring pain associated with swelling and redness of the overlying tissues in its distal third. She had had this complaint for one month and felt that the pain had progressed rapidly in the week prior to being seen.

Examination revealed a thin, elderly white female of approximately her stated age. There was no pathology demonstrated in the examination of the head and neck regions. The heart and lungs were normal to auscultation.

In the lower third of the sternum was a firm, tender tumor measuring 6 x 3 x 2 cms. The skin over the tumefaction was tense, red and shiny.

Abdominal examination was essentially negative. There was an old, well-healed suprapubic incision the result of "having her organs removed" twenty years previously. No masses were palpated.

Pelvic examination revealed a moderate cystocoele and rectocoele. A small atrophied cervix was present with a small erosion of the posterior lip. Bimanual examination failed to reveal any pelvic pathology. The rectum was negative. The cervix was biopsied and Papanicolou smears were taken of

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the cervix and vagina. These studies were subsequently reported negative for tumor.

On 10/14/52 X-ray examination of the lungs and sternum elicited the following information: ". . . the lung fields are clear and well aerated. There is evidence of sclerosis of the aorta. Examination of the sternum shows a loss of bone density in the lower third. This is at the level of the xiphoid process, slightly above the junction of the body. This change is very minimal . . . this area should be rechecked by serial examinations. . ."

The patient was at this point lost to follow-up for approximately 3 weeks before contact was again reestablished. A repeat x-ray examination of the sternum revealed ". . . evidence of destruction of the lower end of the sternum slightly more pronounced than in October. Apparently, there is a soft tissue mass overlying this portion of the sternum. The destruction is not sharp and clear-cut and there is some calcification visible around this area, probably in costal cartilages of the ribs."

On 11/19/52 an incisional biopsy was done. The histological report revealed ". . . clumps of atypical, anaplastic epithelial cells lying in connective tissue stroma. There is a slight resemblance to squamous epithelium in this tissue . . . suggest the possibility of lung as source of the primary tumor."

At this time the patient reported a change in her bowel habits which on previous interrogations had apparently been normal and regular. Sigmoidoscopy revealed no intrinsic pathology in the distal 25 cms. of the colon. On 12/2/52 a barium enema showed an obstructing lesion in the sigmoid. ". . . a rather severe deformity of the lumen was encountered in the middle of the sigmoid loop. This deformity was persistent and . . . indicates malignant involvement of the colon. I cannot determine whether it is primary or secondary."

On 12/15/52 a barium enema study was done again and surprisingly ". . . the sigmoid showed a much better appearance. The obstruction, which was suggested previously, has disappeared. This apparently was an inflammatory lesion of the sigmoid rather than a malignancy." On the same date, a repeat x-ray of the sternum ". . . shows a soft tissue mass surrounding the area of destruction in the sternum. This mass is much more prominent in the lateral view than it was previously indicating growth. . . I do not recognize any distinct pulmonary tumor."

Laboratory studies obtained up to this time were as follows:

RBC 2.35; Hb 7.0; WBC 6,150 with 80% polymorphonuclears. Urinalysis: pH 7.0; S.G. 1.012; alb. slight trace; sugar—negative; microscopic—1 -|- epithelial cells; RBC 0-1, WBC 8-12. Total Proteins, 7.03. A/G ratio, 2.1 — 1. B.U.N. 20.5; fasting blood sugar, 128. Kline—negative Electrocardiogram—within normal limits.

On 12/19/52 cystoscopy was done and a mild cystitis visualized. Retrograde pyelography revealed ". . . the right kidney to be rather large with a large renal pelvis. The right ureter does not show any visible calculous formation. A small uretero-pelvic stricture is suggested on the right side. The left kidney is a double kidney. Two ureters are present almost all the way down to the bladder. A calculous is present in the upper pole of this double kidney."

During the course of the investigations in search of a primary tumor it was thought advisable to irradiate the sternal tumor in order to obtain, if possible, regression of the tumor and some measure of palliation. A full course of external irradiation within the limits of skin tolerance was administered and upon its completion there was a definite improvement in the condition of the patient. She gained weight slowly, became more cheerful and had much less pain. In addition, there occurred a gradual regression in the size of the sternal tumefaction.

At this time the original operative report was uncovered. It noted that " . . . 1½ quarts of free fluid were present in the peritoneal cavity. "Each ovary measured approximately 12 cms. in diameter. A bilateral ovarian carcinoma was present which, in the right ovary, had ruptured. Numerous implantations were present in the base of the pelvis which were responsible for the accumulation of free fluid."

Shortly thereafter, microscopic sections of the original ovarian tumor were obtained and it was the opinion of the pathologist that the tumor histologically, was a cystadenocarcinoma.

On 2/11/53 it was noted on follow-up examination that the patient had developed a large node in her left axilla. This was excised. The histological report follows: "Histological examination reveals lymphoid tissue which has been almost entirely replaced by metastatic tumor composed of cuboidal type epithelial cells varying from one to several layers deep, having large, somewhat atypical nuclei and arranged in a papillary fashion on a fine connective tissue core. Comparison of this tumor with restained sections taken in 1933 from an ovarian tumor present in this patient reveals a marked similarity in appearance. I am now of the opinion that the original lesion in the ovary of this patient is that of a cystadenocarcinoma and that the present material taken from the axilla represents a lymph node metastases from this primary tumor."

The patient, at the present time, feels well, eats well and has no pain. Her sternal tumor has almost completely disappeared.

Although this case represents an unusually bizarre behavior of an ovarian cancer F. W. Stewert recently reported two similar cases that have survived 33 and 35 years respectively.[4]

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BRONCHOGENIC CARCINOMA*
Report of 149 Cases

EMERSON Y. GLEDHILL, M. D.,
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This series of cases represents all the cases of carcinoma of the lung seen and treated on the Tumor Service of the U. S. Public Health Service Hospital, Baltimore, Maryland, from the formation of this Service in 1939 up until July of 1948. At the present time therefore, they represent a determinate five year group of cases. The surgical handling of the cases was in the hands of Dr. William H. Reinhoff, Dr. Ralph R. Braund, and the author. Nine of the total excisions were performed by Dr. Reinhoff and three by the author. All work-up and diagnostic studies were performed by the Tumor Clinic Staff and the cases represent a completely unselected group, 70 of which were locally referred and the remainder transferred from other Public Health Service Hospitals throughout the country. No cases were refused admission for any reason, and terminal care was provided those cases who required it. All cases except one were male, due to the type of patient beneficiary treated by the Public Health Service.

The youngest case was 22 years of age and the oldest 76. The cases were distributed according to decades as follows:

20-30— 1
30-40— 3
40-50—42
50-60—61
60-70—39
70-80— 3

The time period from earliest symptoms to recognition of the need of definitive treatment and admission to our hospital varied from one week to four years, with an average of approximately 9 months. The presenting symptoms of three cases were non-pulmonary and due to intracranial metastatic disease. In one case the initial episode was the finding of a supraclavicular metastatic mass. One case only was completely asymptomatic, which seems unusual in a group of men exposed to regular physical examinations and in most cases regular routine chest roentgenograms.

The carcinoma presented on the right side in 82 cases; on the left in 63 cases; bilaterally in 2 cases; and 2 cases were questionable as to the primary site.

The methods used in securing a proved histological diagnosis consisted of:

1. Bronchoscopic examinations with biopsy or cell examination of smears or bronchial washings.
2. Aspiration Biopsy.
3. Biopsy of Metastatic Lesions.
4. Exploratory Thoracotomy.
5. There were 19 cases in the group that were

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confirmed only at Post-Mortem Examination. One of these cases was unsuspected and considered as a primary brain neoplasm until autopsy.

A total of 102 bronchoscopies were performed on 87 of the 149 cases. These led to a positive diagnosis of carcinoma in 43 cases.

Aspiration biopsy was performed 75 times in 56 cases and yielded positive results in 45 cases. Aspiration biopsy yielded positive results where bronchoscopy was negative in 20 cases.

Bronchoscopic studies yielded positive diagnosis in 49.4% of the cases in which it was used as a diagnostic procedure, while aspiration biopsy yielded an 80.3% positive diagnosis rate. It should be stated however, that in general, aspiration biopsy was used for the most part but not exclusively in those cases with large tumor masses on x-ray examination and those considered probably inoperable.

Biopsy of metastatic lymph nodes or masses was positive in 45 instances and was the only definitive diagnosis in 36 cases.

Exploratory thoracotomy was performed for diagnosis where other methods had failed, in 13 cases.

The histological type of tumor found in this series of cases was as follows:

Epidermoid carcinoma	71
Adenocarcinoma	24
Undifferentiated carcinoma	24
Oat cell carcinoma	12
Anaplastic carcinoma	10
Alveolar carcinoma	2
Transitional cell carcinoma	1
Spindle cell carcinoma	1
No diagnosis	4

The therapeutic approaches used in this series included surgery, x-ray therapy, and a combination of the two. Preoperative x-ray therapy was used in only one instance.

Total number of operations	36
Exploratory Thoracotomy for diagnosis	13
Unresectable tumors	22
Lobectomy	2
Total Pneumonectomy	12

Thirteen cases were given postoperative palliative x-ray therapy. The type of therapy given was 250 kv. opposing port treatments, carrying the tumor dose to approximately 2400 r. in 24 days. In general this type of treatment yielded good results with regard to temporary pain relief and control of hemoptysis and shrinkage of the primary tumor but no cures were recorded. Average length of life following this type of treatment was 7 months, with the longest survival 22 months.

250 kv x-ray therapy as the only type of therapy was used in 89 cases. In these cases there was present conclusive evidence of inoperability. Radon seed implantation was used in only one instance in the treatment of a metastatic lymph node, and once implanted into the primary tumor at the time of thoracotomy.

Total number of cases receiving palliative x-ray therapy as only form of treatment	89
x-ray therapy to primary tumor	81
x-ray therapy to metastatic sites	8

At the present writing, of the 149 cases only two are known to be living and well and free of disease slightly over 5 years from the time of total pneumonectomy by the author.

At the three year period following the completion of the study there were:

131 patients dead
18 living
11 with disease
5 with disease and metastases
2 without disease

Of the patients who died autopsy examination was performed in 85, or 54% of the cases.

SUMMARY AND COMMENTS

This series of cases represents a closely followed-up group treated over approximately a 10 year period and followed for a minimum of 5 years. The final result is very disappointing. Only 2 cases living, well and free of disease after 5 years. Both of these are the result of total pneumonectomy. Earlier case finding, earlier operative intervention, and earlier pneumonectomy with extension of this procedure to include mediastinal lymph node dissection will offer increased hopes in the cure of this disease. Our efforts along those lines should be increased, as statistically this disease is apparently increasing and is now one of the commonest types of cancer.

+ Editorials +

DELAWARE STATE MEDICAL JOURNAL

Owned and published by the Medical Society of Delaware, a scientific society, non-profit corporation. Issued about the twentieth of each month under the supervision of the Committee on Publication.

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Articles are accepted for publication on condition that they are contributed solely to this Journal. Manuscripts must be typewritten, double spaced, with wide margins, and the original copy submitted. Photographs and drawing for illustrations must be carefully marked and show clearly what is intended.

Footnotes and bibliographies should conform to the style of the Quarterly Cumulative Index Medicus, published by the American Medical Association, Chicago.

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Vol. 25 April, 1953 No. 4

Special Session of A. M. A. House of Delegates

The House of Delegates of the A. M. A. was called to a special session in Washington, D. C. on March 14 by the Board of Trustees. The purpose was to consider the President's Reorganization Plan No. 1 of 1953. This plan would elevate the Federal Security Administration to departmental status. The new Department, presumably under Mrs. Oveta Culp Hobby, would be known as the Department of Health, Education and Welfare. Under her would be a Special Assistant who would be "a recognized leader in the medical field with wide non-governmental experience." He would be an M. D. and his duties would include reviewing all the health and medical problems in the Department and advising the Secretary with respect to the improvement of such programs and with respect to necessary legislation in the health and medical fields.

President Eisenhower addressed the House briefly and effectively. He extended a warm welcome and stated that "we have faith that Americans want to do the right thing and that the medical profession will provide the kind of services our country needs better with the cooperation and the friendship of the Administration rather than by its direction or any attempt on its part to be the big 'Pooh-Bah' in this particular field."

Senator Robert Taft then addressed the House. He explained that the various agencies brought into this new Department were all alike in one respect. They were agencies where Federal action and control should be secondary to state and local obligation. He further explained that it should be a Cabinet department rather than a separate agency, because the work should be coordinated with and subordinated to the work of other Departments in the Cabinet. If set up as a separate agency directly under the President there would be a tendency for it to expand out of proportion to its importance in relation to other departments. He said the new department would "build up a philosophy to protect the medical profession for all time to come against intrusion of socialized medicine or anything else compulsory in the American free enterprise system."

Congressman Walter Judd of Minnesota also spoke. He emphasized Senator Taft's remarks in respect to the fact that so many individuals in policy making positions were frozen by civil service and could not now be removed. This made it difficult for the Administration to carry out its plans and promises effectively. They were stymied by workers carried over from the past 20 years. Until many of these individuals are replaced by others willing and anxious to carry out the policies of the new Administration the will of the voters who elected that Administration

could not be carried out. This applied particularly to Mrs. Hobby's agency and the proposed new Department. We were given the assurance that the problem would be met.

Dr. Louis Bauer, President of the A. M. A., then spoke. He stated that for almost 80 years the A. M. A. had advocated an independent Department of Health with an M. D. in the Cabinet. The feeling had recently been modified to the extent that it was felt that the cabinet member need not necessarily be an M. D. There was also some feeling recently that such an independent Department of Health might not be too desirous, after all; one reason being that under an Administration unfriendly to organized medicine it might boomerang. However, the official policy of the A. M. A. has not changed since it was reaffirmed last December in favor of an independent Department of Hea'th. Dr. Bauer stated that this Reorganization Plan, which would unquestionably be passed by Congress in the near future, might be considered as a step toward that goal.

Throughout the session there was evidenced a willingness and desire on the part of the Administration to work with the Association. This was expressed again by Dr. Dwight Murray, Chairman of the Board of Trustees, when he gave the report of the Board on the Reorganization Plan. He stated that for many years the policy of the A. M. A. has almost always had to be one of opposition to the Administration. Now the door is open and we are asked to give to the government our assistance in its medical problems. After a thorough consideration of the Plan, during conferences with the President, Mrs. Hobby and others, the Board of Trustees asked the House of Delegates to support Reorganization Plan No. 1. After a brief discussion the House voted, by unanimous action, to support the Plan.

LAURENCE L. FITCHETT, M. D.,
Delegate to the A. M. A.

MISCELLANEOUS

Have You Paid Your Dues!

The American Medical Association Annual Session will be held in New York City from June 1 to June 5, 1953. The headquarters, Scientific and Technical Exhibits, and other meetings will be at the Grand Central Palace, 44th and Lexington Avenues.

To avoid the usual congestion at the registration windows and to save yourself trouble and delay, the American Medical Association is issuing advance-registration cards which will make it possible to quickly register instead of waiting in line to have your record checked with the membership roster.

To register it is necessary for you to have a pocket card showing your membership dues in the American Medical Association paid for 1953. If you have not already sent in your check to cover 1953 dues, we suggest that you do so at once, so as to give us an opportunity to forward the payment to the A. M. A. in time to have the A. M. A. issue a pocket card and also an advance-registration card to you.

A Statement by Edward A. Rumely

The Supreme Court of the United States has spoken. Once again it upholds Constitutional government. It is now the supreme law of the land that no partisan group, minority or majority, can interfere with the rights of American citizens to print and distribute, and with the rights of American citizens to buy and read what printed matter they choose. This is a new and sweeping victory for freedom of the press.

The decision is as far reaching as the court decision in Colonial times when a jury upheld the right of Peter Zenger, a printer, to question acts of the Crown and its agents.

The Court of Appeals reversed the conviction in the lower court, saying, "Appellant Rumely was within his rights in his refusal to supply the names of purchasers of books.

"But, speech and press, including the new means of mass communication, radio, television and movies *are freedoms protected by the First Amendment.* And the public policy which prohibits any current Congressional membership from abridging the impact of public opinion upon the Congress is as sound today as it was when it was first formulated. If those who today would influence legislation turn from the buttonholes of the legislators to the forum of public opinion for sup-

port, a great good in the cause of representative government has been done."

Had the highest court in the land not upheld my refusal to reveal the names of purchasers of our books, no publisher in the country would have been safe from the possibility of being haled to Washington to reveal his subscription and readership lists.

By its inquisitorial procedure, the Buchanan Committee intimidated prospective purchasers of our books to the extent of several hundreds of thousands of dollars. That sacrifice of legitimate revenue is well worth the loss if it now protects all publishers, private citizens and business firms from any such further inquisition and intimidation.

The Medical Curriculum

Medical educators must restructure the entire medical curriculum to meet the changing health needs of today, according to Dr. George Packer Berry, dean of Harvard Medical School, writing in the March issue of the *Journal of Medical Education.*

Dr. Berry names freedom of opportunity for good health as a basic human right and declares "that this opportunity for good health should be provided for all is an insistent and growing demand."

This movement must come from the medical profession itself and properly starts with the education of future doctors. Dr. Berry goes on to say: "Let us not forget that if we do not do some very hard thinking and acting right now, some one whom we believe to be less competent than we will do the acting for us without thinking. If we believe that we are the best architects, it is up to us to produce the plans."

After outlining some of the plans for active revamping of the medical curriculum now underway, Dr. Berry describes a six-year program of Teaching Institutes to be sponsored by the Association of American Medical Colleges. The purpose of the Teaching Institutes will be to examine the medical curriculum critically to determine what changes need to be made to bring it up-to-date.

Advances in medicine have increased life expectancy by a third since 1900, says Dr. Berry, and in so doing have contributed to the creation of new and difficult problems which medicine must help to solve. Medicine must train men to serve the whole patient rather than merely to cure disease or aid disabilities. This calls for extensive revision of the medical curriculum.

The first such Teaching Institute will be held at Atantic City immediately preceding the 64th annual meeting of the Association of American Medical Colleges October 26-28. The areas covered will be the teaching of physiology, biochemistry and pharmacology. Every medical school in the country will be invited to participate. Future conferences will cover pathology, microbiology, immunology and genetics; anatomy, histology, embryology and anthropology; medical ecology; clinical teaching, including the internship; specialty training and the continuing education of the physician.

Dr. Berry indicates that many medical schools have already undertaken programs to revise their curriculums. He cites Western Reserve University, where a new curriculum was inaugurated this fall and is being watched with interest by medical educators. Other schools mentioned as conducting experiments in their curriculum include Harvard, the University of Tennessee, Boston University, the University of Colorado, and Cornell.

As basic to consideration of curriculum revision, the March issue of MEDICAL EDUCATION also contains a statement, in its eighth revision, of the objectives of undergraduate medical education. The document is the first formal statement of objectives formulated by the Association of American Medical Colleges, and more than 100 educators participated in its development. Broad objectives of medical education are listed as: (1) acquiring requisite knowledge (2) establishing essential habits (3) achieving basic skills (4) developing sound attitudes (5) gaining an understanding of professional and ethical principles.

Most Doctors Don't Want Social Security

Should Social Security be extended to physicians in private practice? Most of these M. D.'s say "No," according to an article in the February issue of Medical Economics, national business magazine for doctors.

The magazine recently asked the question on a survey questionnaire sent to each of its

134,000 physician-readers. Fifty-five per cent of the respondents said they oppose Social Security coverage for private M. D.'s; the remaining 45 per cent said they favor it.

According to the survey, opposition to Social Security coverage is strongest among doctors in small towns, among women doctors, and among doctors in the high-income brackets.

Red Cross First Aid and Water Safety

Because of increased interest in self-preservation and water sports, Red Cross first aid certificates awarded in 1951-52 topped the million mark for the second consecutive year, with 1,090,000; and swimming and lifesaving certificates attained an all-time peak of 813,000. In addition, first aid and water safety demonstrations and talks reached more than 5,700,000 persons.

Average Doctor Has More Patients Now

The average U. S. physician in private practice works a fifty-eight hour week and sees twenty-eight patients in a typical day. His patient load has increased 12 per cent in the past four years.

These figures are revealed in the February issue of *Medical Economics*, national business magazine for doctors. The magazine is publishing the results of a nationwide survey it made recently among its 134,000 M. D.-readers.

Herman E. Gaumer, M.D.

Dr. Herman E. Gaumer, 42, of Bellevue Manor, died April 4, 1953 of a heart attack while playing golf at the Hercules Country Club. Taken to the Memorial Hospital in the ambulance of the Minquas Fire Company, he was pronounced dead on arrival.

With him at the time he was stricken were Dr. Richard E. Chaddock of the Hercules Powder Company, Dr. Olaf P. Bergelin of the Department of Chemical Engineering, University of Delaware, and Dr. Gaumer's 11-year-old son, Richard, who was caddying for the threesome.

Born in Philadelphia in 1910, Dr. Gaumer received his bachelor's degree in 1932 from Pennsylvania State College, where he was a member of the football team and Beta Theta Pi fraternity. In 1936 he received his doctorate in medicine from Hahnemann Medical College, where he was a member of the staffs of Delaware, Memorial, St. Francis and Wilmington General Hospitals and the New Castle County Medical Society, and the Medical Society of Delaware. He was a member of the Presbyterian Church of the Covenant.

An enthusiastic golfer, he was a member of the Concord Country Club and the Kennet Square Country Club.

During World War II he was a captain in the Medical Corps of the Army, and he saw service in the European Theatre of Operations.

Surviving, in addition to his son, are his wife, Mrs. Elizabeth Dilsworth Gaumer; a daughter, Judith, 11; his parents, Mr. and Mrs. Herman Gaumer Sr. of Philadelphia, and three brothers, Albert and Elmer S., both of Philadelphia, and John L. Gaumer of Blowing Rock, North Carolina.

Funeral services were held on April 7 at the Presbyterian Church of the Covenant, Bellevue, the Rev. Dr. Gordon R. Coning, minister of the church, officiating and interment was in Lower Brandywine Cemetery.

BOOK REVIEW

PLASTIC AND RECONSTRUCTIVE SURGERY—A Manual of Management. By Ferris Smith, M.D., Consultant in Plastic Surgery, Blodgett Memorial Hospital, Grand Rapids, Michigan. Pg. 895, with 592 figures. Cloth. Price, $15.00. Philadelphia: W. B. Saunders Company, 1950.

Dr. Smith's book is an excellent adjunct to the teaching material used in the preparation of an individual in plastic surgery. It, by itself, is not presented so as to teach the general or orthopedic surgeon to be versed in the field of major reconstructive procedures. Its use will mainly come to those already versed in plastic and reconstructive surgery, or those already training within that specialty.

The first portion of the book is devoted to the general principles of plastic surgery and is very well presented. This section is recommended to all those in any field of surgery.

The main portion of the book is concerned with reconstructive procedures of the head and neck.

The concluding part discusses reconstructive procedures of the extremities.

The bibliography appended to the end of the book is good, as is the format, etc. This book should be in the library of the hospital as well as in the library of any plastic surgeon.

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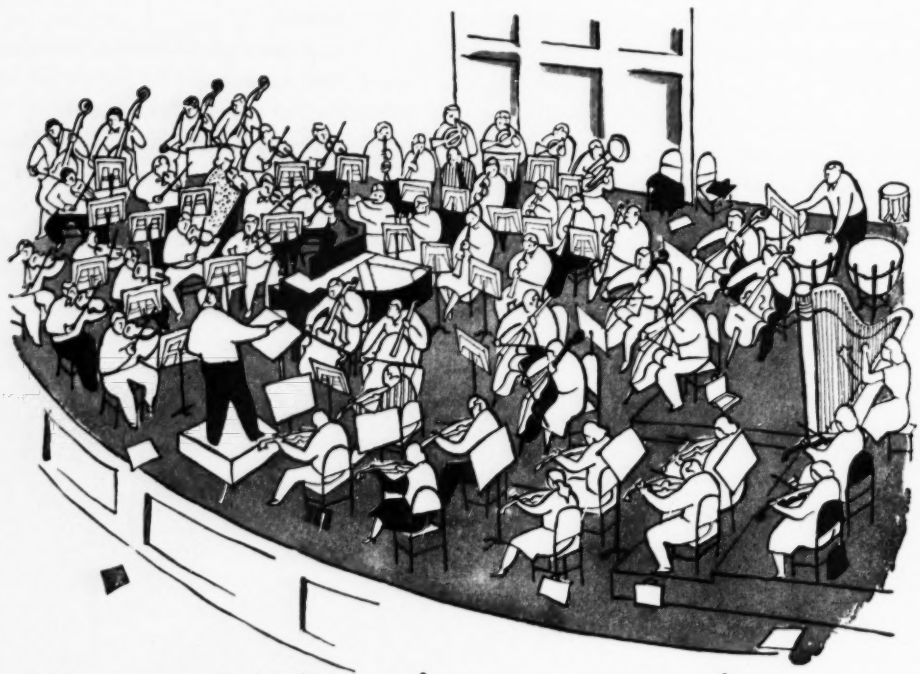

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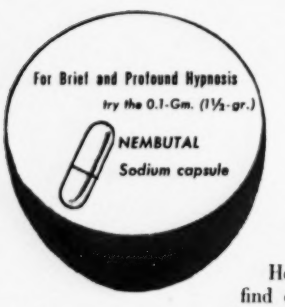

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Operating Room in Hospital	10.00	20.00	30.00	40.00
Anesthetic in Hospital	10.00	20.00	30.00	40.00
X-Ray in Hospital	10.00	20.00	30.00	40.00
Medicines in Hospital	10.00	20.00	30.00	40.00
Ambulance to or from Hospital	10.00	20.00	30.00	40.00
COSTS (Quarterly)				
Adult	2.50	5.00	7.50	10.00
Child to age 19	1.50	3.00	4.50	6.00
Child over age 19	2.50	5.00	7.50	10.00

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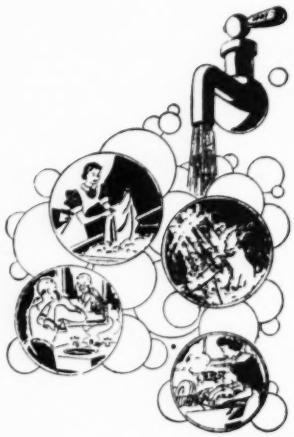

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1. Frost, L. H., and Jackson, R. L.: J. Pediat. 39: 585-592, 1951.

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